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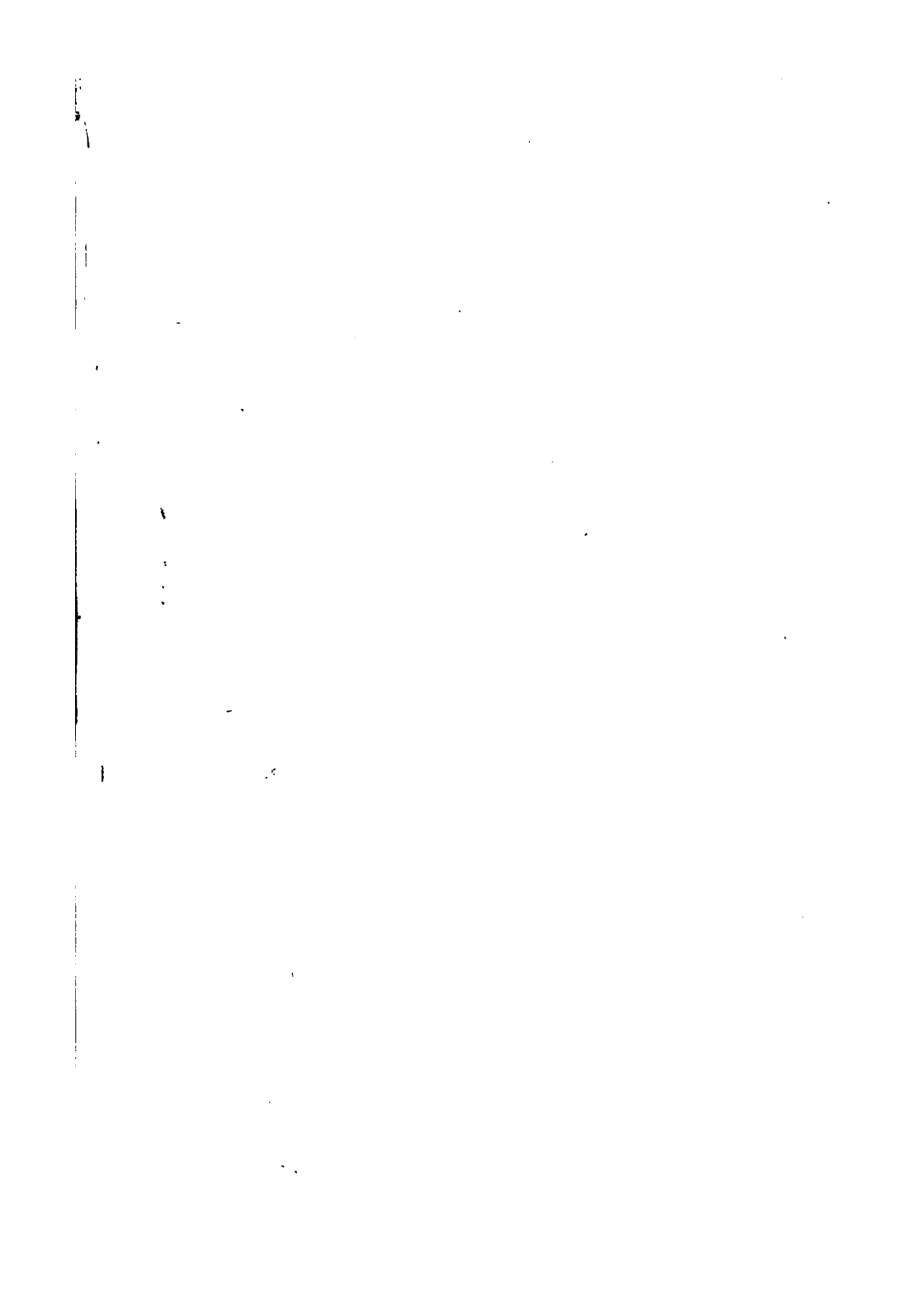


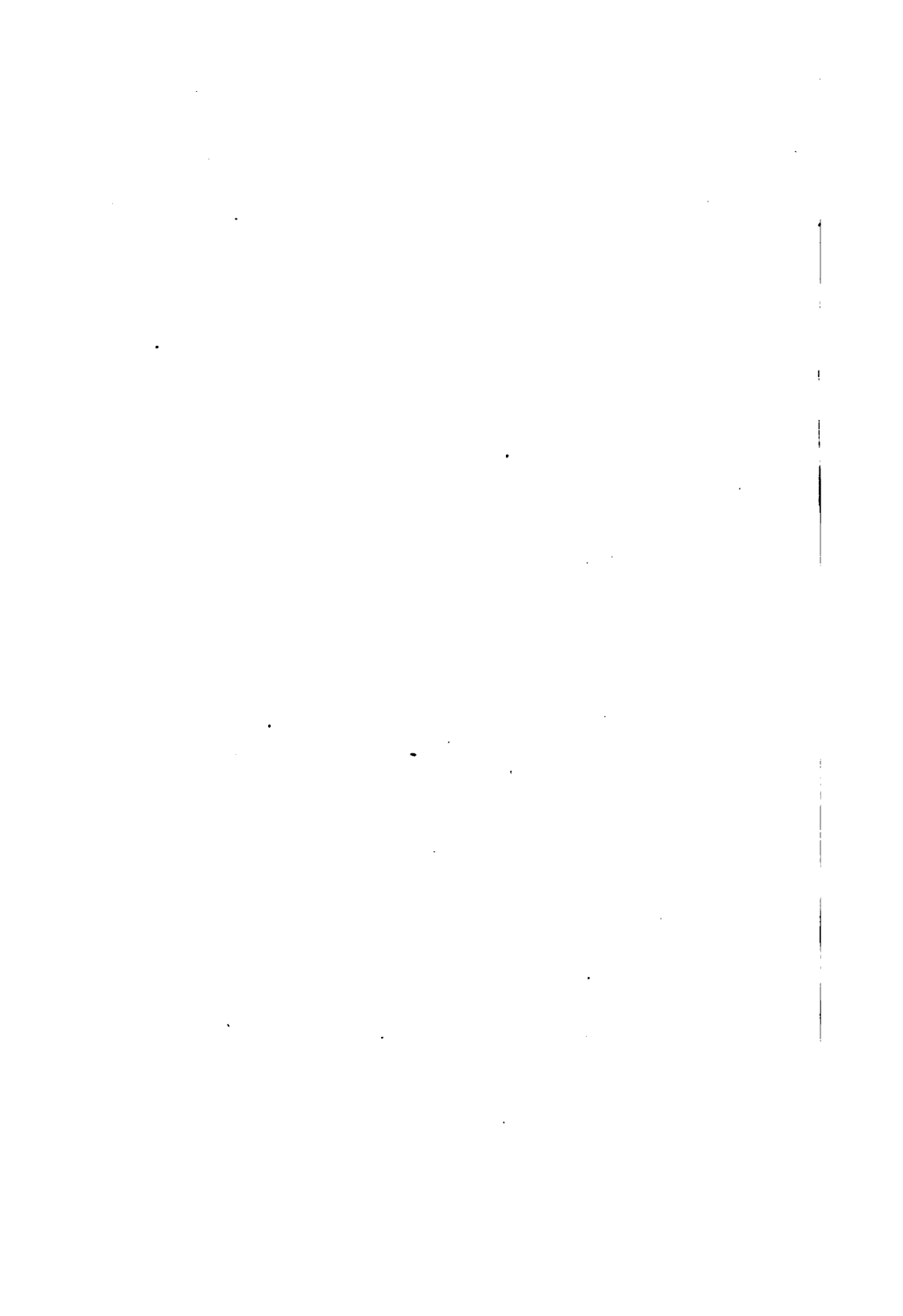
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VOLUME X

**NERVOUS AND MENTAL DISEASES**

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**SERIES 1916**

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CHICAGO

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## DISEASES OF THE NERVOUS SYSTEM.

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### SYMPTOMATOLOGY.

**The Value and Meaning of the Adductor Responses of the Leg.** A. Myerson<sup>1</sup> reports a study of a series of periosteal reflexes invoked by percussion of the bones of the lower extremities and marked by the response of adductor muscles. His experience has led him to conclude that adductor responses are present in health as mild and occasional homo-lateral and contra-lateral responses from the internal condyle, from the middle of the shaft of the tibia, from the internal maleolus and from the Achilles tendon. Other sites, such as the sole of the foot, the patellar tendon, the external surface of the knee-joint and the anterior superior spine of the ilium may be the starting points of adductor responses. Fatigue does not increase these responses, but diminishes them to the point of abolition. In general paresis, the adductor responses are very prominent, especially in the early stages of the disease. When the knee-jerk has disappeared or is diminished in tabes the adductors disappear. In hemiplegia due to cerebral hemorrhage, thrombus or embolism on the side of the paralysis the adductor reflexes are livelier than on the opposite side. In certain diseases such as multiple sclerosis and compression of the cord, which are associated with increased knee-jerks, the adductor responses follow the same general principle as do the knee-jerks. In dementia praecox, in general, the adductor responses are not conspicuous. The above is true of manic-depressive insanity. In hysteria the homo-laterals are frequently lively; the contra-laterals not so conspicuous. As a result of his experience, Myerson believes that the adductor responses, whether

(1) Jour. Nerv. and Ment. Dis., February, 1916.

homo-lateral or contra-lateral, elicited from the patellar tendon, the anterior superior spine and the external condyle, practically exclude neurasthenia as a diagnosis.

In his *résumé* he states:

"The appearance of contra-lateral adductor responses, especially from the patellar tendon, the external condyle or the anterior superior spine, is usually a sign of disease and frequently of organic disease. The responses appear to have some relationship to the knee-jerk on the side of the muscles responding, but not to the knee-jerk on the side stimulated." He points out that the site of stimulation is probably not so important as the direction of the blow and the resultant stimulation of either hip-joint or pelvis, and that the part thus indirectly stimulated acts as the afferent limb of the reflex arc, the motor limb of which leads to the adductors. The adductor muscles probably belong to the tonic group of muscles and receive their innervation from sources other than the cerebrum. With the disappearance or diminution of cerebral influence, the tonus of these muscles is so increased that their reflex activity becomes greatly enhanced.

**The Clinical Value of Defense and Muscle Reflexes. Importance of the Latter in Spinal Localization.** As to the value of defense reflexes, T. H. Weisenburg<sup>2</sup> says that from an investigation made, which included all types of spinal-cord and brain lesions but in all of which there was pyramidal involvement, he can not say that the presence or absence of the pathologic reflexes of defense had any distinct diagnostic value. He could not come to the conclusion that the pathologic defensive reflexes indicated an irritation or compression of the cord. In some spinal-cord cases, especially in those in which there was flexor contracture, the defensive movements were quite marked but on the other hand when they were obtained they were at times equally marked in the extensor types of paraplegia. The impression gained was that the more severe the stimulation the greater the defensive movements. For example, it was a common experience when irritating the soles of the feet or the legs that at first no defense movement

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(2) Jour. Nerv. and Ment. Dis., September, 1916.

would be obtained, but repeated stimulation would after a while cause prompt defensive movements. Occasionally pin irritation produced defensive movements when pressure and pinching would not.

There are undoubtedly exceptional cases of spinal-cord disease such as Babinski has described in which there is great increase of tonicity with exaggerated tendon reflexes and with at times permanent extension of the large toe in which defensive movements can be obtained by stimulation of the skin up to the area corresponding to the lower limit of the lesion. But these cases are necessarily rare, and we must finally conclude that in the usual run of spinal-cord cases the pathologic reflexes of defense are not of distinct diagnostic importance.

The pathologic reflexes of defense must not be distinguished as distinct from the dorsal extension of the large toe. Dejerine and Mouzon, for example, in reporting various types of spinal cord lesions following war injuries designated the defensive reflexes as contractions of the tensor fascia lata, adductors and the sartorius, but not including the dorsal flexion of the large toe. This was not the experience of Weisenburg and his colleagues, for it was impressed upon them that in no case did they obtain a dorsal movement of the large toe without a simultaneous movement of some of the muscles of the thigh.

The most productive part, however, of the investigation concerned the muscle reflexes. By a muscle reflex is meant the response of either the whole or part of a muscle when tapping the body of the muscle itself. This of course is present in a normal individual but the response is not marked. In every instance in which the motor columns are diseased the muscle jerks are very prominent. For example, in hemiplegia the muscle jerks are increased on both sides, but they are distinctly more so on the hemiplegic side. In all cases of myelitis with the exception of those instances in which there is complete destruction of the cord, the muscle jerks are always exaggerated up to the upper limit of the lesion. In the peripheral segments corresponding to a destructive process in the cords the muscle reflexes

are either diminished or absent. In cases of tabes the muscle jerks are diminished, while in poliomyelitis they are absent in the diseased areas. The intensity of the responses depends on the hypertonicity of the muscles. The greater the hypertonicity the more marked the response.

Of course muscle irritability is increased in functional diseases such as hysteria or neurasthenia or in diseases such as tetanus, but in such instances the important point is that the muscle jerks are prominent in all parts of the body.

For diagnostic purposes then the value of the muscle reflexes consists either in their diminution or absence in limited areas or in their increase in limited areas.

Weisenburg's conclusions follow:

1. Exaggerated muscle reflexes are constantly present in pyramidal lesions and are of equal importance with increased tonicity and increased tendon reflexes.

2. In spinal localization they are of value, for in destructive lesions they are either absent or diminished in the peripheral area corresponding to the lesion. In such cases the muscle jerks may be increased just above the lesion but are always exaggerated in the parts of the body below the lower limit of the lesion, even though the skin and tendon reflexes are diminished or lost.

3. In incomplete lesions of the spinal cord the muscle reflexes are exaggerated up to the upper limit of the lesion.

4. In a gradually extending upward paralysis such as is produced by a pressure myelitis the exaggeration of the muscle reflexes keeps pace with the increase of the paralysis and is an indication of an extension of the lesion.

5. Finally the most important conclusion that can be drawn is that the exaggeration and more especially the absence of a muscle reflex is an indication of the state of the reflex arc in the spinal segment corresponding to that particular muscle.

[It also must be noted that care must be exercised in differentiating the increase of direct myotatic irritability as seen at some stages of peripheral motor neuron lesions, wasting diseases, fevers, etc.—Ed.]

**Babinski's Sign from the Point of View of Comparative Anatomy.** In a series of papers published in the *Russian Journal of Psychiatry, Petrograd*, M. Ast-wazaturof<sup>3</sup> has brought forward arguments showing that in the explanation of symptoms observed in lesions of the pyramidal tracts it is necessary to call attention to some facts of comparative anatomy.

He continues: "To say that the extensor plantar reflex is a spinal reflex, while the normal plantar reflex is a cortical one, is no solution of the problem. In fact, the sign of Babinski is somewhat enigmatic; it is neither an exaggeration nor an abolition of a function, but an inversion of it. We must search for some special mechanisms which, being latent in the normal subject, are set in action by a lesion of the pyramidal tract—that is, when the communication between the spinal cord and cerebrum is interrupted.

"What is the nature of these mechanisms? A reply to this question is hinted at by the fact that the extensor reaction of the great toe is quite a normal phenomenon in infants who have not learned to walk. Thus the sign of Babinski appears to be a normal phenomenon during a period of the ontogenetic evolution of man.

"In a lesion of the pyramidal fibers—that is, in a case of separation of the propriospinal apparatus from the ne-encephalic elements of the cortex:

"1. The voluntary impulse can not reach the periphery, whence arises paralysis or paresis.

"2. The inhibitory influence of the cortex on the propriospinal apparatus ceases—thence appear: (a) the increase of reflexes, the clonus; (b) the propagation of the reflex irritation from segment to segment in form of "*reflexe de defense*" and analogous symptoms (the pandiculation of Bertolotti, femoral reflex of Remak, etc.)

"3. Lastly, there arise some reflex reactions corresponding to some earlier stage of phylogenetic evolution. These reactions are nothing but effects of mechanisms latent in normal cases and manifesting themselves in the case of loss of the cortical influence on the spinal cord.

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(3) Abst., Austral. Med. Jour., August, 1916.

"And so Babinski's sign comes into existence; the normal flexion of the great toe is neither exaggerated nor diminished after a lesion of the pyramidal tract, but is inverted into an opposite reaction of the extensor type.

"So in pyramidal lesions, the impulses provoked by irritation of the sole must spread to the centers to which these impulses used to spread when the function of the foot was a grasping one. Thus they spread to the flexors of the second, third, fourth and fifth toes. As for the great toe, its opponens muscle, to which in this stage of phylogeny have spread the impulses from the sole, is lost in man, therefore there would be no reaction of the great toe."

**Syncope.** Lovell Lanstroth<sup>4</sup> reviews the causes of syncope. He thinks that most probably loss of consciousness is due to a sudden diminution of blood-pressure within the skull and thus, indirectly, to a diminution in the blood-supply of the brain. He refers to the experiments of Kussmaul and Tenner, who produced unconsciousness by compression of the carotids in a number of male adults and to the production of unilateral convulsions by compression of a single carotid by Leonard Hill. The vagus is given a prominent place as a causative factor of certain syncopal attacks. Thalhoffer observed unconsciousness in a student who made pressure over his own vagi. Syncope is common in diseases of the heart and vessels, in aortic disease, during the compensatory phase of a ventricular extrasystole, a slow sinus rhythm, paroxysmal tachycardia and fibrillation, in paroxysmal congenital cyanosis, aneurysm of the arch of the aorta and in heart-block.

[This is of some interest relative to the cause of unconsciousness in epilepsy where it has been observed that preceding a *petit mal* attack a sudden drop of blood-pressure occurs. The relation of the vagus to such syncopal attacks is likewise of great importance when viewed along with the facts that certain medullary convulsants (picrotoxin, coriamyrtin) are central autonomic nervous system poisons, and the frequency of vagal and autonomic symptoms in epilepsy and its attacks, the aura

(4) Amer. Jour. Med. Sci., January, 1916.

vascular and respiratory symptoms associated with the attacks, salivation and contracture of the vesical musculature must all be considered.—Ed.]

**False Innervation.** H. Oppenheim<sup>5</sup> says that the conception of false innervation, i. e., an unphysiologic and improper distribution of innervation impulses, is seen in ataxia, apraxia, asynergy, etc. He calls attention to still another group in which, in injuries of the peripheral nerves, the energy of moving certain muscle groups sets into action still other muscles, as in attempting forcibly to close the fist, contractions occur in the abductors of the upper arm, triceps and platysma muscles. At other times the false innervation is limited to antagonistic muscles, which receive so increased an innervation that the movement "willed" produces an opposite effect. In case of a severe gunshot injury of the radial nerve with complete paralysis, the patient could not extend his hand, whereas he could support it forcibly if passively extended first. In the effort to extend it the flexors were so strongly innervated that the effort was fruitless.

Oppenheim noticed a similar phenomenon in cases of paralysis of the trapezius and serratus magnus where the rhomboids were falsely innervated, and in injuries of the ischiatic nerve where upon attempts to flex the foot dorsally contraction of the quadriceps occurred. He warns against the misinterpretation of this phenomenon as hysterical manifestation.

## THE NEUROSES.

### EPILEPSY.

**A Classification of the Epilepsies.** Feeling that there is a pressing need for a new classification of the epilepsies, Elias C. Fischbein<sup>6</sup> offers a classification which is constituted upon clinico-symptomatologic lines, the object being, so far as possible, to define each group sharply, to give it a name that will refer to characteristics which are constant and peculiar to that group, so

(5) Neurol. Centralbl., November, 1915.

(6) Medical Record, Sept. 2, 1916.

that the name will be not only designatory in a convention sense but also descriptively correct. An endeavor has been made to correlate these names so that each may suggest its antithesis.

Explaining the scheme, he says of some of the groups that seem unfamiliar and need elucidation.

*Paroxysmi Inter Bibenda* (convulsions while drinking; "rum fits"): Attacks occurring only when the patient resorts to drinking.

*Paroxysmi in Alcoholophilia* (convulsions occurring in a person exhibiting alcohol-desire): In other words, epilepsy occurring in a drinker.

*Paroxysmus Infantum Eccentricus* (reflex infantile convulsion): The infant may be of normal physical and mental makeup, but there may occur temporary reflex disturbance of the very sensitive nervous organism. Such disturbance may be caused by gastro-intestinal disorder, fevers which involve the brain and its matrices, diarrhea resulting from dentition or worms, etc. A type which is manifested by hyperexcitability of the peripheral nervous system, resulting in tendency to tonic and clonic spasms (chorea, laryngismus stridulus, tetany, apnea, carpopedal contractions, etc.). To this condition has been given the name "Spasmophilia" or the spasmophilic diathesis. Phenomena coming under the group of spasmophilias should be distinguished from true epileptic paroxysms, as they have not been proven to be strictly epileptic in nature.

*Paroxysmi Parturientium* (puerperal convulsions; eclampsia gravidarum): Common major motor seizures upon a uremic basis.

*Paroxysmi Uraemici* (uremic convulsions): At times these attacks can not be distinguished symptomatically from the common major motor seizures. They occur in nephritis with its concomitant symptoms. There is usually no initial cry. The onset is sudden. Convulsions may occur every hour or two and there is deep coma between attacks. Following such seizure or series of attacks there may be coma persisting for several days or weeks. There may be amaurosis without visible retinal lesion and there may be development of monoplegia or diplegia.

*Myoclonus* (paramyoclonus multiplex of Friedreich; choreic tics; multiple tics; myoclonus spinalis multiplex; myokymia): This term, which is in common use, meets our needs. It so happens that myoclonus and its modified forms is one of the few syndromes properly cognomenated. Briefly the distinguishing features of this affection are lightning-like contractions of groups of muscles. The contractions may involve only one group of muscles or the whole body. Consciousness is not lost. They may occur as epiphenomena to major or minor seizures, or as an uncombined symptom-complex. These contractions may also involve one group of muscles, later extending to the whole body musculature and, in certain instances, terminate in a major motor seizure.

*Pseudomyoclonus*: Single or multiple myospasms on a hysteric or choreic basis. The name has also been applied to the sudden localized muscular contractions seen immediately before major motor seizures, but these must be regarded as part of the aura.

*Tetanilla* (tetany) is mentioned here only as a reminder that it occurs in relation with epilepsy and often requires diagnostic consideration.

*Hemicrania* (megrim; migraine; cephalalgia; neuralgia cerebialis): This type of nervous disturbance occurs either in relation to a seizure, as an equivalent or unaccompanied by attacks. The headache, although termed "hemicrania," never occurs as exactly confined to one side; but it is usually felt as more severe on one side than on the other. Migraine resembles epilepsy in several ways. Both are hereditary. Both are periodic in point of occurrence. Both may show an aura. Migraine may be replaced by a psychic equivalent, like any epileptic attack. Both migraine and epileptic seizures are followed by somnolence.

*Narcolepsia Hysterica* (hysteroid narcolepsy): This form may occur in epileptics in the form of status catalepticus, or in non-epileptics in the form of trance. Therefore we have:

(a) *Status Catalepticus* (catalepsy, stupor vigilans, catochus, etc.). A hysterical form of narcolepsy observed in epileptics. Hysteroid in nature and probably not dependent on the condition causing the epilepsy.

In this occurrence there is a sudden suspension of the action of the senses and of consciousness with great muscular plasticity, the limbs and trunk preserving the different positions given to them. A rather rare condition.

(b) *Catalepsia Hysterica* (trance; ectasis): A condition which is purely hysterical and not in relation with epilepsy. It consists of sleep episodes which last for days and weeks. It is interpolated here for differentiation.

Of dual personality, Fischbein says that it should be noted in the discussion of this type, and that while these episodes occur in persons who are neuropathically tainted and who may later develop epilepsy, still they may occur in people who are not epileptics.

[The various groups here defined are largely such as are not ordinarily included in our conception of epilepsy. The inclusion of many conditions which are of hysterical nature seems to us to add to the possibility of reopening the hazy fusion of such non-existent conditions as hystero-epilepsy, etc.—Ed.]

**Epilepsy with Olfactory Aura.** H. Mauris' reports a tumor of the brain in a man of 37, who after an illness of two months, which was ushered in by a severe epileptic attack, died following a second attack. These attacks were on both occasions preceded and followed by olfactory hallucinations of sweet, sickly and disagreeable character. The olfactory sense otherwise was normal. At autopsy a tumor of the right uncus of the gyrus hippocampus was found. This involved the larger part of the gyri hippocampus, lingualis and fusiformis, together with the whole Ammon's formation.

Mauris says that for the production of an epileptic attack it is necessary to irritate an aura center. In this case the olfactory was the center involved.

**Syphilis in Epilepsy.** William T. Shanahan, J. F. Munson and A. L. Shaw<sup>8</sup> state that the failure of specific treatment to cure their patients and the lack of specific lesions in a few cases of supposed syphilitic epi-

(7) *Zeitschr. f. d. ges. Neurol. u. Psychiat.*, November, 1915.

(8) *New York Med. Jour.*, April 29, 1916.

lepsy that have come to autopsy, have raised the question in their minds of the real importance of syphilis in the production of epilepsies.

The problems they attempted to solve are: (1) The occurrence of syphilis in epileptics; (2) the etiologic relationship of syphilis to epilepsy; (3) the results of treatment; and (4) the existence of a special type of epilepsy due to syphilis.

In reviewing the literature on the occurrence of syphilis among epileptics they state that Veit gives 7 per cent. of hereditary lues among the patients of the institution at Wuhlgarten, Germany, and Bratz, from the same institution, found syphilis in the parents of 5 per cent. of 400 patients. Noguchi reports on fifty-one cases in which syphilis was not ascertainable, finding 20 per cent. of positive and 6 per cent. of doubtful reactions in the blood serum. In the cerebrospinal fluids of these patients, he obtained 4 per cent. positive and 2 per cent. of doubtful reactions. Pleocytosis was absent in all.

Alden Turner saw only four cases of congenital lues among 1,000 cases of idiopathic epilepsy, and these presented no unusual features. He also saw one case develop during the secondary state of the disease. Osler says that convulsive seizures due to acquired syphilis of the brain are very common. In the Massachusetts institution for epileptics, Wassermann reactions in 535 consecutive cases showed 3.34 per cent. of positives. Walker and Haller found 14 per cent. of seventy-one cases of epilepsy without other apparent cause, to give positive Wassermann reactions. Of their ten positive cases, three patients have had no attacks for a year after treatment with salvarsan; in the remaining seven, treated with mercury intramuscularly, the attacks were relieved while under observation. In thirty-five of these cases, including the ten positives, the cerebrospinal fluid was Wassermann negative and gave a normal cell count.

The clinical material of the Craig Colony for Epileptics amounts to approximately 4,100 cases, and by gathering data from all sources, there appear to be 133 cases in which syphilis was suspected, or 3.2 per cent.

These group themselves as follows:

	Cases.
Diagnosis doubtful .....	4
Unknown if hereditary or acquired.....	33
Hereditary cases .....	54
Acquired before epilepsy.....	27
Acquired after epilepsy.....	6
Relation of syphilis to epilepsy unknown.....	9

It might be stated that clinically what has been termed syphilitic epilepsy does not present an entity of symptoms of any diagnostic value, except possibly where apoplexy occurs in a patient during the third or fourth decade of life with perhaps a resulting hemiplegia and recurring convulsions, either local or general in type.

While it might seem that in the cases in which epilepsy is apparently the result of syphilis specific treatment would be beneficial, in the majority of these cases material damage has already been done to the brain and its membranes, so that it is ordinarily futile to apply specific treatment with the expectation of effects beneficial to the epilepsy.

They conclude: "An epileptic with syphilis is not necessarily a patient with syphilitic epilepsy.

"The occurrence of syphilis among our patients does not seem to differ much from the occurrence of syphilis among the population as a whole.

"Luetin tests show the highest proportion of positive tests in our cases, while the Wassermann positive cases are somewhat below 2 per cent. Wassermann reactions on the cerebrospinal fluid would probably give a still lower figure.

"Treatment is unsatisfactory, owing probably to the lateness of its beginning.

"In many cases the diagnosis is exceedingly doubtful and should be supplemented by examination of the patient's parents, brothers, and sisters, not forgetting the use of the Wassermann and luetin tests.

"There is no type of syphilitic epilepsy.

"Syphilis is one of the many agencies which produce the disorder we call epilepsy."

**Blood Findings in Epilepsy.** Ralph H. Spangler\*

(9) Lancet, April 29, 1916.

reports on the examination of 100 epileptic patients. The hemoglobin is not reduced by epilepsy itself. The erythrocytes showed an average of 4,640,037. About one-tenth of the cases showed more or less crenation of the red cells, but only in 5 per cent. of the cases were there present a large number of poikilocytes or microcytes. The leukocytes ranged between 3,650 and 9,360 during the interparoxysmal periods. Shortly after attacks they varied from 5,062 to 37,550.

As a general rule Spangler's experience would indicate that an epileptic attack is almost invariably followed by temporary leukocytosis. The differential count indicated an increase of large lymphocytes, in 98 per cent., and in small lymphocytes in 51 per cent. In only 2 per cent. of the cases did the clotting time during the interparoxysmal period exceed the normal average of 5 minutes and 6 seconds. In 98 per cent. of the cases it was below the normal average, and in 52 per cent. it fell below the shortest limit of what is considered the normal range (from 3 to 8 minutes). An investigation of the alkalinity of the blood of twenty-four cases with the Dare method indicates: (1) The daily average of alkalinity of an epileptic's blood is invariably below that of a control individual using the same diet. (2) An epileptic attack causes no further diminution of the alkalinity of the blood, nor does the severity of the convulsion (muscular exertion) have any modifying influence. (3) A night and morning estimate of the alkalinity for a month, during which period both major and minor seizures occurred, failed to demonstrate any noteworthy modification of the alkalinity either before or after either form of seizure.

**Blood Cultures in Epilepsy.** William B. Wherry and Wade W. Oliver<sup>1</sup> report that in cultures from the blood of six patients with epilepsy they have failed to isolate the organism described by C. A. L. Reed as the *Bacillus epilepticus*.

H. Caro and D. A. Thorn<sup>2</sup> report that:

1. In a series of seventy cases, with a total of 160

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(1) Jour. Amer. Med. Ass'n., Oct. 7, 1916.

(2) Idem.

blood cultures, there were 156 sterile cultures. The remaining four showed contaminations.

2. Four cases with either myoclonus or hemiplegia also gave sterile blood cultures.

3. In a series of seventeen necropsies on epileptics, Dr. Canavan, in a bacteriologic study, was unable to find any organism resembling *Bacillus epilepticus*.

4. In conclusion, it would seem evident that, in the seventy epileptic patients studied, the epileptic syndrome was not due to the *Bacillus epilepticus* of Reed.

Referring to the records of six cases, Charles A. L. Reed<sup>3</sup> says:

"It will be seen from an analysis of these records that positive findings were not made in every specimen taken. In the files of Dr. Hyatt's reports now before me, I find several cases in which positive findings were not reached until in one instance after the sixth. In easily one-half of our cases, the finding was negative at the first examination. As nearly as I can estimate in this record, the ratio of positives to negatives is as 7:13. In leafing through this file and taking the cases as they come alphabetically, I find one series of five and another of eight, in which the first examinations proved negative. It will be seen from this that the negative findings of Wherry and Oliver in single examination of four cases and three examinations of one case are not the least surprising, especially in view of the fact that one of these cases did prove positive in their own medium. The same observation applies with even greater emphasis to the report of Caro and Thom."

After investigating the results of Reed,<sup>4</sup> W. B. Terhune<sup>5</sup> summarizes his work as follows:

Cultures were made from twenty-four epileptics during or immediately following seizures; from eighteen in whom no convulsions had occurred for several days; and from forty-two non-epileptics. He says.

"Although I began this work profoundly biased in my opinion, as I believed that epilepsy was not bacterial in origin, I am forced to the conclusion that the bacillus which I have isolated, which is identical with the *Bacil-*

(3) Jour. Amer. Med. Ass'n., Oct. 14, 1916.

(4) Pract. Med. Series, 1916, Vol. X, p. 28.

(5) Jour. Amer. Med. Ass'n., Oct. 14, 1916.

*lus epilepticus* described by Reed, must be an etiologic factor in epilepsy, in view of the following facts:

"A bacillus was isolated from 75 per cent. of epileptics examined.

"It was present during and following a seizure but not during the intraconvulsive period, except in the case of one patient who was debilitated by ill health.

"It was not found in non-epileptics.

"It causes typical epileptoid convulsions in cats during which death occurs, both when they are injected intravenously and when they are fed cultures of the organism.

"The organism may be recovered from the animal during the convulsions and after death."

**Calcium and Epilepsy.** Guy P. V. Prior and S. Evan Jones<sup>6</sup> report the result of investigations made on the calcium blood content and the excretion in the urine of epileptics, together with clinical observations on treating epileptics with salts of calcium and with extracts of those ductless glands that control the calcium metabolism. They examined the blood and urine of thirty-five patients, and compared them with similar examinations made on twelve members of the staff. In the case of the staff, the average blood content was within the normal range and the average urine content normal; whereas the blood content of the epileptics was 0.6 of normal and the urine content 0.14 of normal.

Although their work is still tentative, and though their results are imperfect and fragmentary, and have not extended as yet over a sufficient number of cases or length of time to enable them to draw definite conclusions. They think their investigations tend to show:

1. A diminished amount of free calcium in the blood of epileptics, also a diminution of the excretion in the urine.

2. That epileptics may be benefited by administration of calcium. In the case of two patients who were treated for a period by calcium alone, the number of fits in each case fell below the previously recorded minimum.

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(6) Med. Jour. Austral., March 4, 1916.

3. The effect of calcium is great, if combined with bromides.

4. Calcium and bromides, given together, give better results than either calcium or bromides alone.

5. Menstruation precipitates fits by promoting calcium changes; and by increasing the calcium index by hypodermic injections the number of fits has been diminished.

During the period under review, the total number of attacks of all the patients was less than average for a similar period by 528: a reduction of 31 per cent.

**Treatment of Epilepsy.** Francis X. Dercum<sup>7</sup> in describing the treatment of epilepsy says that the first indication is that the organism, though defective and deviate, shall lead as physiologic a life as is compatible with its structure. To attain this end, a life without physical or mental strain, close to Nature, in a camp or on the farm, should be adopted by the epileptic. This, indeed, is the principle applied in the various epileptic colonies. In a given number of cases it is attended by an improvement in general health and a notable diminution in the number of seizures. There can be no doubt that the benefit is largely due to the increased oxidation of waste and toxic substances and the general increase of physiologic efficiency which result from an outdoor life. In addition, says Dercum, three points should be borne in mind:

"1. The diet should be so modified that in this organism, already toxic, as little strain as possible be placed on the liver, the thyroid and other defensive glands. For this reason the red meats are to be partaken of sparingly. The carbohydrates also are to be diminished. To take the latter in large amount is to hamper the oxidation of the tissues, an oxidation which for the obvious reason of the autotoxicity of the patient should be maintained at as high a level as possible. In the diet, emphasis should be laid on the white meats, the succulent vegetables and milk; eggs also may be permitted. Stimulants of all kinds are, of course, to be excluded.

"2. The various avenues of elimination should be kept

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(7) Jour. Amer. Med. Ass'n., July 22, 1916.

freely open. If the diet does not of itself counteract the constipation frequently present, a moderate dose of a simple saline or laxative water may be given daily. The patient should drink water freely between meals to promote the action of the kidneys, and should take a lukewarm sponge bath daily to promote the action of the skin. The bath should not be such as to promote an active reaction, but merely to favor elimination.

"3. Resort to medicines must, of course, be had in many cases to influence or control the seizures. Time will not permit the extended discussion of these, but after all is said and done experience teaches that chief reliance must be placed on the bromides. Regarding their efficient administration, however, one important point must be borne in mind, namely, the principle of sodium chloride withdrawal introduced by Richet and Toulouse. If table salt is withheld, the bromides instead of being eliminated are retained and are effective in such smaller dose. I myself have been in the habit for many years past of administering the bromides in the form of sodium bromide, at the same time instituting as rigid a withdrawal of the sodium chloride as possible. There can be no doubt that under these circumstances the sodium bromide takes the place, in a measure, of the sodium chloride in the tissues. If, in a case so treated, the sodium bromide be discontinued and sodium chloride resumed, the bromide is rapidly eliminated in the urine.

"But one other point of importance remains. In a given number of instances, the physiologic level of the patient may be distinctly raised by the administration from time to time of small doses of thyroid extract; say from  $\frac{1}{8}$  to  $\frac{1}{4}$  grain three times daily, seldom more. Thyroid in small doses, long continued, stimulates the chain of glands of internal secretion, increases oxidation and promotes metabolism generally."

#### HYSTERIA.

**Mechanism of Hysterical Phenomena.** In preparing a theory for the explanation of hysteria, A. Salmon<sup>8</sup>

(8) Jour. Ment. Sci., April, 1916.

refers to the curious phenomenon which he described at the Congress of Neurology in Florence. This is exactly the condition mentioned below as being designated by Rothman as *Nach Bewegung Phenomenen*. Salmon concludes that this act is automatic and is dependent on the residue of muscular, articular and tendinous sensations which are produced after the muscular contraction, in other words, to their kinesthetic usages. He sums up as follows: "I am convinced that hysteria finds its clearest explanation in the study of kinesthesia. Just as this function elucidates the mechanism of all our voluntary and automatic actions, in the same way it explains the mechanism of hysterical actions which are affected according to the laws which regulate voluntary and automatic acts. The fundamental condition of the affection is, in my opinion, a hyperesthesia or an active impressionability of the kinesthetic usages, endowed with a strong affective and motor tendency, which, attracting to themselves all the nervous dynamic energy, bring about a disequilibrium of kinesthesia, and consequently modifications of the personality peculiar to hysterics."

By this theory, Salmon explains the capability which hysterical troubles possess, created by emotions and affective ideas of repeating themselves independently of these latter. Similarly, the relation between organic lesions and hysteria, and the impulsive and plastic power of effective ideas; in a word, it seems to Salmon that his theory "is not in contradiction to the principle theories which have been advanced in the study of this malady, but that it clears up the most obscure points in these theories, thus rendering them easier of acceptance."

**The "Nach Bewegung" Phenomenon. (Katatonic Experiment of Kohnstamm.)** This phenomenon consists of an after-contraction or movement which appears after long continued contraction of muscle groups against resistance, as may be seen by pressing forcibly the extended lowered arm and forearm, held near the body, against a wall for a period of from thirty to sixty seconds. After a short latent period the arm spontaneously raises itself. Csiky<sup>9</sup> obtained this phenomena after a

(9) Neurol. Centralbl., October, 1916.

faradic tetanus and concluded that it was an idio-muscular contraction, which originating in the fatigued muscles extends itself to the whole muscle mass.

**Hysterical Mutism.** Dr. G. Hudson Makuen<sup>1</sup> describes hysterical mutism as a form of mutism by no means common, bearing a close resemblance to the other manifestations of the disease, occasionally existing quite independently of all other hysterical symptoms, and differing from other forms of mutism in that it is of the profoundest character.

While Dr. Makuen has seen but two well defined cases of hysterical mutism, seventy-one cases were collected by Marcel Natier of Paris more than twenty-five years ago. Dr. Makuen's first case was reported before the American Laryngological Association in 1906 and was one of so-called traumatic hysteria in which the hysterical symptoms resisting all the known methods of relief yielded to an emotional excitement of an explosive nervous attack in the form of a nightmare or hysterical fit, during which the patient uttered several sentences with normal and phonatory and articulatory precision. Following this attack, speech had remained normal for the several years he had been under observation. The second case was in a girl of 13, who had always been more or less subjective minded and melancholy. Three years previously she had had four convulsive seizures, preceded for several months by failing health and with some loss of power in the limbs, presumably the result of severe fright. The mutism was complete and the futility of attempting to cure her in her home environment being evident, she was about to be placed in a hospital when she was taken by friends to a religious camp-meeting and induced to "pray back" her voice. The enthusiasm and excitement were said to have been intense and the girl was urged to pray long and loud. Apparently this was the process necessary to bring about a mobilization of the child's psychical forces enabling her to speak with a laryngeal sound after many months of complete aphonia. While probably contended by those healing in this way that the cure was purely in answer to prayer, Makuen concedes that the exercise of

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(1) New York Med. Jour., May 27, 1916.

faith is doubtless an important factor in the psychic change.

He concludes that:

Hysterical mutism is a somewhat rare affection, but it occurs at all ages, in all races, in both sexes, and even in animals.

Hysterical mutism is usually of sudden origin and the result of some severe psychical or emotional shock.

Hysterical mutism is characterized by the complete absence of the psychical representations necessary for the production of speech.

Heredity is an etiologic factor so far as it furnishes the neuropathic or psychopathic soil for the development of the affection.

The mutism generally occurs in conjunction with other hysterical phenomena, although it may be the only hysterical symptom.

Hysterical mutism may occur in conjunction with organic nervous diseases which themselves simulate hysteria, and it can be distinguished from them only by actual discovery of the pathologic conditions giving rise to the particular affection."

Arthur J. Hall<sup>2</sup> reports a case of mutism occurring in a boy 11 years old.

This case seems in some respects to form a link between the hysterical mutism of peace and the mutism occurring under war conditions. The boy, although not in the trenches, was exposed for a short time to terrific shell fire at Hartlepool. Six months later he was in close proximity to an explosion which may fairly be compared in severity to that of a trench mine. Yet neither of these tremendous shocks was sufficient in his case to produce immediate "mutism." It was reserved for an ordinary surgical operation of everyday occurrence in civil life to supply the back-breaking straw, and determine the onset of symptoms.

As regards the determination as to what form the hysterical manifestation takes, it is interesting to note that in this boy the mutism followed an operation on the throat—the neighborhood of voice production. So

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(2) *Lancet*, May 20, 1916.

also, ordinary hysterical aphonia is often preceded by laryngeal catarrh.

It does not seem unreasonable to suppose that, in cases of mutism following shell explosion, the particular form which the hysterical manifestation assumes is determined by the powerful effect necessarily produced upon the auditory apparatus, which, as we know, is intimately associated with the spoken word.

Recovery in this case was sudden and dramatic. The ward in which the boy was confined was an adult male medical ward; at one end there is a small single ward. The occupant of this single ward was an old man, of somewhat wild appearance, with long hair standing straight up from his head. During the early hours of the morning this old man wished to go to the closets, which are at the opposite end of the ward. He therefore put on his dressing-gown and walked down the ward. As he came round the broad pillar of the central fireplace of the ward the little boy happened to wake up, caught sight of this bizarre figure in the semi-darkness, and screamed. The old man, who had heard of the arrival of a dumb boy, and wishing to be friendly, came up to the bed and asked him if he were the "dumb boy." This intended kindness frightened the boy still further. The result was that he shouted for the nurse. The spell was broken!

#### CHOREA.

**Treatment of Chorea.** Morris Grossman<sup>3</sup> from a study of fifty cases describes a method of treating chorea by means of re-educational exercises. These consist of breathing, relaxation and re-education exercises. During the acute stage breathing and relaxation are alone used.

The abdominal type of breathing is insisted on; the patient is asked to take a deep breath, using his diaphragm, restricting his thoracic movements, and at the height of inspiration to pause, then slowly and evenly expire, and again pause. This deep breathing soon tires the patient if persisted in, so after ten or twelve of these

(3) New York Med. Jour., May 27, 1916.

deep respirations have been taken, the depth of inspiration and the pauses are shortened until the patient is breathing, without effort, as in sleep.

To relax the muscles, passive movements in which the muscles are alternately lengthened and shortened are employed. The muscles of the forehead, cheek, and jaw are thus manipulated until wrinkling of the forehead, and blinking of the eyelids disappears and muscular spasm is eliminated. Next a shoulder is relaxed, then an arm; each in turn must be passively moved until all traces of muscular tension vanish and the part lies motionless and flaccid, and falls limply from any unsupported position. Then the leg on the same side should be taken. After a part is relaxed, those previously and that newly relaxed should be briefly dealt with again, in the order in which they were first relaxed.

Movements should be begun after the patient has thoroughly mastered relaxation and his limbs can be freely moved passively in all directions without exciting either rigidity or spasm. Simple movements only should at first be used—flexion, extension, adduction, and abduction at the joints; the movements should be guided along the proper plane by the operator, until the incoördination has been corrected. The movements should then be done by the operator with the patient offering slight resistance; the patient next does the movement unguided, and finally the movements are performed against the resistance of the operator. The movements should be of the simplest character at first and gradually made more elaborate as the patient improves.

The movements should be executed slowly and steadily, and without jerks or flourishes; during every movement the patient counts rhythmically—the purpose of the counting is to educate him to move easily, uniformly and at regular tempo.

Grossman concludes that:

1. Relaxation and re-education have a distinct value in treatment of chorea.

2. The course is favorably modified and the danger of complication lessened; the duration of the disease is shortened by their use.

3. Residual chorea and other conditions of muscular unrest are obviated.

4. We reduce to a minimum the drain on the motor centers, due to prolonged and continuous muscular activity.

5. With this systematic exercise treatment, we increase the mental stability and will power of the patient, thereby diminishing the danger of subsequent ties and other functional neuroses, that flourish in such a soil.

**Hospital Treatment of Chorea.** Pearce Bailey<sup>4</sup> in detailing the treatment of chorea emphasizes the importance of rest and isolation.

This means rest in bed, with curtains drawn, and no communications allowed with other patients in the ward and no visitors. In certain cases, cold packs are given, and in the presence of rheumatic history, and even without it, rheumatic remedies, especially aspirin, are prescribed. In a few violent cases Bailey has used lumbar puncture, which, when the cerebrospinal fluid is under increased pressure, seems to diminish the movements very promptly. In view, however, of the long time the disease has existed before the patient enters a hospital and the violence of the motor restlessness, it seems probable that several weeks' complete rest is necessary to overcome the irritability of the nervous system which must have been engendered, so that even if after a lumbar puncture all the symptoms disappear, it seems to him wiser to insist, whenever possible, on a three or four weeks' treatment for the purpose of re-establishing the tone of the nervous system. He has rarely found it necessary to give salvarsan.

Most of the patients who stay in the hospital less than ten days leave for some reason other than that directly connected with the disease. In a few patients in whom choreic manifestations disappear in two or three days, the parents have taken the children home. Bailey does not feel, however, that ten days is sufficient time for the treatment of even mild chorea. The period between ten and thirty days is the one in which most of the cases fall. At the end of this period in the majority of cases

(4) New York Med. Jour., Sept. 23, 1916.

no twitchings have been noticeable for some time, and the patient, so far as can be seen, is well, except for cardiac murmurs or lesions which may still persist. A few patients who have stayed longer than thirty days have either had cases of very long standing, or else are those in whom the chorea seemed merely another manifestation of a deeply imbedded long standing rheumatic tendency. Sleep after the first few days is generally very good, although some restlessness and talking may persist for a longer period.

The author has been unable to draw inferences from the condition of nutrition. Some patients who do not do particularly well, gain in weight; others in whom the twitchings disappear rapidly, lose weight, but in the majority there is no material change in weight during the stay in the hospital.

His general conclusions from cases of chorea treated in the hospital are that by rest and seclusion, when the treatment is extended over a reasonable period, chorea can be permanently cured without much danger of a relapse, and in view of the general relapsing tendency of the disease, feels that the treatment of chorea is one of the most important functions of a neurologic hospital.

**Marinesco's Method.** From the results of this form of treatment in seven cases, and from those of other authors, Augusta Natali<sup>5</sup> draws the following conclusions:

1. Intraspinal injections of magnesium sulphate in the proportions and doses indicated by Marinesco, give good results in cases of chorea minor.

2. There is always an improvement in cases of chorea minor; at times soon after the first injection, at others sometime afterwards. In some cases improvement is progressive up to complete recovery; in others a second or even third injection is necessary before any improvement can be noticed or a cure effected.

3. Recurrences are rare.

4. A short arsenical treatment is of value following the treatment in question.

5. The method of Marinesco is not to be used in all cases of chorea, as the percentage of cures or improve-

(5) Amer. Med., August, 1916.

ment is not greater than that obtained by the use of other drugs, but should be restricted to two classes of cases: (a) cases in which other treatment has not given good results; (b) cases in which the choreic movements are pronounced to the point of interfering with the patient's rest and therefore need immediate reduction.

**Treatment by Lumbar Puncture.** At a meeting of the New York Neurological Society, Walter Timme<sup>6</sup> reported three cases of chorea treated by lumbar puncture and stated that the results at the New York Neurological Institute tend to prove the benefits to be derived from this method. On the other hand, Abrahamson warned against the indiscriminate performance of lumbar puncture inasmuch as he had noted a case which was made much worse by the procedure.

#### OTHER NEUROSES.

**The Factor of Fear in Nervous Cases.** In an article filled with clear clinical examples of the factor of fear in functional nervous diseases, H. T. Patrick<sup>7</sup> says: "We fear the unfamiliar; the things of which we are ignorant." All his life one of his patients had been used to tough men with pistols but not to physicians' instruments. "German students are timid about bare knuckles but not a bit afraid of the *Schlager* that hacks their faces to pieces. The boldest financier may be inexpressibly scared about his liver. The one almost universal fear of the human race is Hades, or what follows death—the one thing of which we all know absolutely nothing.

"But to this rule there are notable exceptions: exceptions which show what a fundamental and powerful emotion fear is, what a primitive race we are and how we still are largely controlled by instincts and emotions; not by knowledge and judgment."

To state the thesis another way, says Patrick, intellectuality seems to be no effective protection against phobias. It is largely a question of temperament. The intellectual and cultivated are quite as susceptible as the stupid and ignorant. And regarding this temperament

(6) Jour. Nerv. and Ment. Dis., August, 1916.

(7) Jour. Amer. Med. Ass'n., July 15, 1916.

one may frequently make a most interesting observation of what at first sight looks like a psychologic paradox. Concerning these phobia patients one very often hears members of the family say, "She never gives up," "Nothing discourages him," "He's a hard fighter," "She's full of courage and patience," and the like. All of which is perfectly true, and yet this person is in the clutches of an overpowering fear. The explanation lies in the nature of the individual. They are the people dominated by an idea.

The born enthusiast makes a fine phobic. Had her obsession taken another direction, Joan of Arc would have made a beautiful case of *folie du doute* or of agoraphobia. Martyrs and profound psychasthenics are often made of the same stuff.

To insist that one should be careful not to confuse phobia with impulse, the fear of doing a thing with a sudden desire to do it, would seem to be a work of supererogation. But patients themselves frequently make this mistake, and physicians sometimes follow their example. One of the best English text-books on insanity cites in some detail a typical case of phobias as one of homicidal impulse.

A frequent fear, which the patient looks on as an impulse, is that of doing some *mal à propos* thing in public; of using obscene language or yelling or slapping someone or making a suggestive movement. Frequently such fears are really fear of insanity, of suddenly losing self-control. But the conception of the dreaded act recurs so constantly and forcibly that the patient gets the idea that he has a tendency to commit it, and the doctor takes his word for it. Needless to say, the diagnosis is vital for both prognosis and treatment, but is generally very easy. The patient at once acknowledges that he does not want to do the thing, never did want to do it, and has no reason for doing it, but fears that for some reason in some way he may suddenly have an abnormal impulse to do it, and will not be able to resist the impulse.

Properly to elucidate a case of phobia, manifest or latent, generally one must go back to the very first appearance of the symptoms and investigate the circumstances surrounding it. In most instances the mechanism

is simple. The patient has had some distressing or peculiar sensation or some disability from adequate physical cause. This sensation he at once or (more often) subsequently correlates with some serious malady or calamity which in some way has been brought to his attention. It goes without saying that the patient has as prerequisite a hypersensitiveness or impressionability. Consequently, often a very small trauma suffices. From his cases, Patrick notes as the initial disturbance: ptomaine poisoning, tobacco poisoning (excessive smoking), dizziness or syncope from over-heated and over-crowded rooms, alcohol poisoning, the weakness or trembling from typhoid or other acute illness or following confinement or an operation, heat-stroke, discomfort from excessive eating, rheumatism, aural vertigo (quite a number), tinnitus, syphilis, nocturnal emissions, the shock and hemorrhage of initial coitus, cerebral thrombosis, migraine, the distress of hyperacidity or indigestion, uremic convulsions, night numbness or night palsy, and predormal shocks.

In considering the treatment of these cases, Patrick says:

“First: The remedies most frequently prescribed for these disorders are absolutely futile except for possible suggestive value. How can ‘tonics’ or ‘sedatives,’ change of climate, a vacation, tacking up a floating kidney, lifting up a prolapsed uterus or rest in bed and massage eradicate fear? Would a winter in Florida, a trip around the world, an operation for hemorrhoids, or strychnine pills make a sinner less afraid of eternal punishment?

“Second: The way to remove fear is to show the patient what it is and then teach him to demonstrate to himself that it is groundless. In short, educate him out of it as he has been inducted into it. If a child is afraid of a dark room we do not give him soothing syrup and tell him there is no room and no darkness. Nor do we suddenly throw him into the place of dread. We explain the darkness and the entire absence of anything to be feared. Perhaps we light the room and then make it dark again. Then, when we are sure he is ready for it, we take him kindly but firmly by the hand and lead him into the dark room, or perhaps only to the door or part

way in. Finally, he goes in alone, and when he has fully demonstrated to himself that there is nothing there to be feared, of course he is not afraid and he is cured. We are only children of a larger growth. And not so very much larger either."

**Treatment of Neurasthenia.** Austin Fox Riggs<sup>s</sup> says that as distinguished from the psychasthenic, the neurasthenic is simply the type that is annoyed and needlessly incapacitated by his exaggerated sensations, and therefore refers his symptoms to his body.

The psychasthenic, on the other hand, though belonging to the same general category, is too acutely aware of his mental processes, is annoyed by his emotions, and therefore refers his symptoms to his mind.

The hypochondriacal variety seems to Riggs to be "an exactly similar breed of cat," and may belong to either type, but with the worry and fear element sufficiently predominating to change the clinical picture—but only superficially.

Studying the statistics of his cases, he finds nervousness in one or both parents occurs in 52.4 per cent.; nervousness + insanity, in 2.7 per cent.; + alcohol or drugs, in 0.3 per cent.; + constitutional diseases, in 6.9 per cent.

A study of the occupations brings to light nothing of interest—not even a suspicious absence of occupation, much as this fact would disappoint the railers against "nervous prosperity."

So far as sex goes, in his practice the women lead the men in the proportion of three to one.

As to age, neurasthenia, like other forms of functional disorder, and by this he means disorders without gross or discoverable lesion, occurs in late adolescence and early maturity with greater frequency. Of his patients, 56 per cent. were above 20 and under 50 years old, the largest number, 33 per cent., being between 30 and 40, when they applied for treatment.

A very frequently recurring factor among the possibly predisposing causes is the personality of the patient. Something like 80 per cent. of them speak of their "temperaments" as causal. "Sensitive" or "high-strung"

are the terms most commonly used by them and their friends, and Riggs thinks in the main that they are right, for temperament, *i. e.*, personality, is at least one clew to the situation.

So it is at least a fair assumption, that the parental neurasthenia has had a great, if not the only, effect on most cases as an environmental element.

His conclusion is that: Neurasthenia is primarily a mental disorder; that the disorder, in nearly all cases, is originally independent of any and all bodily conditions, and that it exists, of course, in spite of a structurally normal central nervous system.

Were neurasthenia exhaustion, rest would cure it. It does not; in 49 per cent. of the cases which this report covers, the patients had received definite "rest cures" but still remained neurasthenic.

Were it an inherent weakness of the organism, not a single patient could be cured. Many are cured.

Were it due to physical disorders, then physical treatment of these disorders would cure it.

The conclusion seems clear, he asserts, that neurasthenia is not weakness or exhaustion; that it is neither a malady of the intestines, the heart, or the stomach, nor a disorder dependent on structural change of the nervous system; and that it, therefore, can not be cured either by rest or by any other physical means.

He has found in psycho-analysis, when guarded, a valuable diagnostic tool, especially in that most important matter, the study of personality; and it frequently either blazes the way for active therapeutics or guides it on its way.

"Fundamental re-education of the patient is the weapon in which I put my greatest trust. To teach the neurasthenic what neurasthenia is, to teach him to contrast this state of mind with the normal, to show him that he can attain this normality, is, I believe, 75 per cent. of curing him. The other 25 per cent. consists in applying this knowledge to practice.

"Coupled with the re-educational psychotherapy there must, of course, be rational physical treatment, based on the particular need of each patient. This part of the treatment is carried out according to a definite schedule

for each patient, in which exercise plays an important rôle and includes individual calisthenics, as well as out-of-door walks, golf, skating, or snow-shoeing; while rest plays a distinctly subordinate part, and is given no physiologic importance whatever.

"Only for complete physiologic bankrupts should rest be at all an important item and, even in these cases, I have never continued it at an aggregate of over three hours daily, exclusive of the night, for more than a week or ten days. Rests, though short, should be made as perfect in quality as possible; that is, patients should be taught how to rest.

"This part of re-education is especially important for those who suffer from the syndrome called insomnia, which is no more, no less, than a fear of not sleeping, a nocturnal apprehensive restlessness, which is the cause, not the effect, of the sleeplessness.

"Rests, then, are considered rather as object lessons in re-education, illustrating what non-interference and non-responsibility will do, than as important physiologic items.

"Occupation, both manual and intellectual, I consider one of the most important instruments of re-education, for through it one can teach efficiency more objectively and more directly than, perhaps, by any other method. For, by studying a patient's way of working, one can actually see the faults in expenditure of energy and correct them, so to speak, on the spot.

"We find that weaving for women and wood-carving for men answer the purpose admirably. Intellectual occupation, on the other hand, is varied more according to each patient's personal resources and needs. Reading, coupled with analytical summarizing, is a frequent prescription, the work being preferably on a subject closely allied to the patient's normal job.

"As I have said, re-education is the method on which we place our greatest reliance. This is carried on through a series of office visits, which are never hurried, and therefore frequently pretty long—they average an hour, I should think. At first, until the patient is well started on his schedule, a daily visit seems best. Then one every two or three days is sufficient.

"A thorough physical examination precedes treatment. I mention this, not because of the obvious fact that it is an essential preliminary to any treatment, but because I consider it the fundamental starting-point of the therapeutics of re-education, especially from the patient's point of view. A pupil must know that his teacher is himself personally familiar with the facts, otherwise he can not be expected to have any real confidence in the teaching. This examination is also purposely and patently made the direct basis of the physical regimen prescribed, and it thus serves another obvious therapeutic purpose.

"The relation of teacher and pupil being at least fairly well established, re-education proper is begun by a few explanatory talks on normal physiologic psychology, without at first any reference to the patient's difficulties.

"These talks deal with the nervous system reduced to a very simple diagrammatic form, in which it is pictured as a telephone system with the brain as central office, while consciousness is considered as made up of energies transformed and liberated by this mechanism.

"As soon as the patients have acquired a good working modicum of self-control, and their physical condition has become satisfactory—with as little warning before hand as possible—they are sent home on a trial trip, to apply their knowledge and the fruits of their practice to their own environment. Their stay at home varies from two and three weeks to six months or more in length—according to the case and the environment. A return for treatment usually follows the trial trip. It is in most cases short—a few days only—and usually consists in a critical review of the successes and failures in adjustment, and other experiences, brought out by the trial trip. The next home-going is apt to be permanent, but is often followed in six months or a year by another and final visit of a few days."

## THE CEREBROSPINAL FLUID AND DISEASES OF THE MENINGES.

**Reaction of the Cerebrospinal Fluid.** S. H. Hurwitz and C. L. Trauter<sup>1</sup> state that the colorimetric method of determining the hydrogen ion concentration of the cerebrospinal fluid gives constant and reliable results. The method, when applied to cerebrospinal fluid, possesses greater accuracy than in the case of other biologic fluids, notably blood. The simplicity of the technique makes it applicable as a routine procedure in the examination of spinal fluids.

As determined colorimetrically, normal cerebrospinal fluid is more alkaline than blood, the difference in the hydrogen ion concentration of the dialysates of the two fluids being equal to 0.45, the value of pH for cerebrospinal fluid being 8.11; value of pH for blood, 7.66. No alteration from the normal reaction has been noted either in the blood or in the fluid of patients suffering from primary or secondary syphilis or from syphilitic affections of the nervous system. So far no study has been made of the reaction of the cerebrospinal fluid in acute inflammatory conditions of the meninges. The demonstration that a change in reaction does or does not occur would have an important bearing upon the value of hexamethylenamine as a therapeutic agent in the prophylaxis and treatment of meningeal infections.

**The Spinal Fluid Syndromes of Nonne and Their Diagnostic Significance.** In 1903 G. Froin reported three cases which upon lumbar puncture showed spinal fluids yellow in color (xanthochromia) containing numerous cells, and which, upon standing, coagulated spontaneously and massively, owing to their high fibrin content. The phenomena thus described were new to medical literature, and they have since been collectively designated "Froin's syndrome."

Some five years after Froin's publication, M. Nonne reported three cases of cord tumor, the spinal fluids of which contained an excess of proteid (strongly positive Phase I) with no leukocytosis (pleocytosis) of the fluids.

(1) *Archiv., Int., Med.*, June, 1916.

In a communication, Frederic M. Hanes<sup>2</sup> brings forward evidence both from his own experience and the literature, with the object of proving that the syndrome of Nonne is simply the early manifestation of a process which in its later and terminal phases gives rise to the syndrome of Froin.

Discussing the differentiation between erythrochromia and xanthochromia he says that the two conditions possess certain characteristics which permit of their ready differentiation:

1. The color in erythrochromia varies from a bright red through varying shades of reddish-brown, reddish-yellow to a dark yellow color. Lumbar punctures made at intervals of several days in a case of hemorrhagic spinal fluid have shown a variety of color changes (Schwarz), whereas the color in xanthochromatic fluids remains the same from puncture to puncture. The shades in xanthochromia are described as amber, cream, or straw colored, and instances are recorded in which the color remained the same throughout several months.

2. In erythrochromatic fluids red blood cells or their shadows may be very numerous. In xanthochromia the white cells are, as a rule, not increased; there are no red cells, and the fluid does not yield either chemical or spectroscopic evidence of hemoglobin derivatives. When the white cells are increased it is evidence of meningeal inflammation.

3. The fibrin content of xanthochromatic fluids is extremely high. The citron-yellow fluid, which is quite limpid, coagulates spontaneously, and in so massive a manner that the containing test tube can be inverted without loss of its contents. The coagulum is gray or white. This is not true of erythrochromatic fluids. They may contain fibrin to a certain extent, but they do not coagulate massively.

4. Both xanthochromatic and erythrochromatic spinal fluids contain proteid substances in very large amount, but whereas in xanthochromia a large excess of proteid is a constant and characteristic part of the picture, in erythrochromia the proteid tends to decrease in amount

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(2) Amer. Jour. Med. Sci., July, 1916.

the further away in time from the hemorrhage the fluid is removed.

The syndrome of xanthochromia with massive coagulation of the spinal fluid and high proteid content, with or without pleocytosis, is always produced by a localized obliteration of the pia-arachnoid space which divides it into two parts, an upper one in free communication with the pia-arachnoid spaces of the upper cord and brain and a lower cul-de-sac. In this lower cul-de-sac the fluid gradually changes in character from the normal limpid spinal fluid to the xanthochromatic type of fluid just described.

He remarks that proteid increase without pleocytosis has the same significance in the early stage of cord compression that the full-blown xanthochromia syndrome has in later stage of more or less complete paraplegia. Indeed, the stage of xanthochromia with massive coagulation may never be reached, and only the early phase of proteid excess without pleocytosis exist as evidence of cord compression.

In conclusion he says:

1. Compression of the spinal cord and its meninges from whatsoever cause leads to the formation of a cul-de-sac, more or less complete, distal to the site of compression. This leads to characteristic changes in the spinal fluid.

2. The earliest characteristic change has been described by Nonne as an increase of proteid (Phase I positive) without cell increase (pleocytosis).

3. As the condition of cord compression persists, the fluid gradually becomes yellow in color (xanthochromia), the proteid content increases enormously, and the fluid, when removed, coagulates spontaneously (Froin's syndrome). Pleocytosis may or may not be present, depending on whether or not the meninges are inflamed by the pathologic process causing the compression.

4. Xanthochromia of the spinal fluid must be distinguished from staining of the fluid by hemoglobin derivatives (erythrochromia).

5. The spinal-fluid syndrome of Nonne-Froin is very helpful and reliable in the diagnosis of spinal-cord

lesions. When present it always indicates a compressive lesion of the cord.<sup>3</sup>

**A Practical Method for Estimating the Protein Content of Cerebrospinal Fluid.** J. A. F. Pfeiffer<sup>4</sup> states that sulphosalicylic acid has been shown to be an excellent recipient for quantitative work, and has been utilized by him for some time in the routine examination of the cerebrospinal fluid for protein in the following manner, which has proved to be a most convenient, practical and satisfactory method: Three cubic centimeters of a 4 per cent. solution of sulphosalicylic acid are placed in a test tube 1 cm. in diameter, and 0.5 c.c. of cerebrospinal fluid allowed to flow down the side of the tube on to the surface of the acid, when a characteristic white ring appears, if an excess of protein be present, which reaches its greatest intensity in two minutes. An approximate estimation as to the degree of excess may be obtained by performing the test in several tubes, with dilutions of the spinal fluid from one to five, and comparing the results.

**Potassium Permanganate in Examination of Cerebrospinal Fluid.** From an examination of over 100 specimens of spinal fluid, A. I. Rubenstone<sup>5</sup> finds the spinal fluid test, advocated by Boveri, very useful: To 1 c.c. of cerebrospinal fluid in a test tube, add 1 c.c. of 0.1 per 1,000 of potassium permanganate, and shake well. Pathologic spinal fluid produces a bright yellow color, while normal fluid retains a violet tinge. He concludes that "while the potassium permanganate test is not of specific value in the diagnosis of pathologic conditions of the spinal fluid, yet we feel that it ought to take its place as an addition to our list of reactions, because it is not only useful in corroborating other chemical tests of the severity of the disease process, but in doubtful cases it may serve as a deciding factor in the differentiation between absolutely normal and mildly abnormal fluids."

(3) Xanthochromia of the cerebrospinal fluid was discussed in Practical Medicine Series, 1912, Vol. 10, p. 6. A lengthy review of all reported cases of massive coagulation (Froin's syndrome) by C. L. Mix is found in "The Surgical Clinics of J. B. Murphy," 1915.

(4) Med. Record, Jan. 8, 1916.

(5) New York Med. Jour., Nov. 20, 1915.

W. O. Hoffman and A. B. Schwartz<sup>6</sup> determined the permanganate reduction index of Mayerhofer on the spinal fluids obtained from patients admitted to the hospital for a number of different conditions. The amount of decinormal permanganate solution which, boiled for ten minutes in a strongly acid medium, is reduced by 1 c.c. of cerebrospinal fluid, is called by Mayerhofer the "permanganate reduction index."

They conclude that indices classify themselves into (1) low indices, below 2; (2) borderline indices, between 2 and 2.5; (3) high indices, above 2.5.

All normal fluids or fluids obtained from patients presenting convulsions or other meningeal symptoms without actual inflammation of the brain or meninges give low indices. Such an index may be obtained in brain tumor.

Borderline indices may occur in the early stage of an inflammatory process, involving the brain or meninges, in serous meningitis, encephalitis or other conditions associated with hyperemia of the brain. High indices, if constant, almost invariably indicate an actual inflammatory process of the brain or meninges. The two diseases most likely occurring with such indices are tuberculous meningitis and acute poliomyelitis. The Mayerhofer test, the authors state, possesses a distinct value in the examination of the cerebrospinal fluid.

**The Sugar Content of the Spinal Fluid in Meningitis and Other Diseases.** Arthur H. Hopkins<sup>7</sup> points out that although mucinoid matter, pyrocatechin, etc., may be present in the spinal fluid, the reducing substance is chiefly glucose.

In regard to the relation between blood sugar and the sugar content of the spinal fluid, using Bang's method, he has found that in health the concentration in the blood averages 10 mgm. per 100 c.c. higher than that of the fluid. In meningitis he has shown that the blood sugar is considerably increased, whereas the sugar content of the fluid is materially decreased.

From the prognostic standpoint it has been found that as the bacteria lose their virulence and their ability to break up sugar and as they become more difficult to culti-

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(6) Archiv. Int. Med., Feb. 15, 1916.

(7) Amer. Jour. Med. Sci., December, 1915.

vate from the fluid, the sugar content gradually increases and is in turn followed by convalescence. This increase may be ascertained by repeated lumbar punctures even before clinical improvement has been observed, and hence its value as the earliest sign of improvement.

A series of diabetics showed a striking increase in the sugar content of the fluid, which, however, usually remained lower than that of the blood.

In a short series of injections the values lie approximately within normal limits.

Among the conclusions he says: "The reducing substance of the fluid is frequently increased in uremia, a condition, however, in which hyperglycemia also occurs.

"A slight increase in the sugar concentration of both the blood and spinal fluid occurs in some cases of epilepsy, as it does in certain other nervous conditions, but the variety of cases observed in this series of thirty-three cases, and consequently the limited number of any one disease, renders inadequate any conclusion drawn at this time.

"Syphilis frequently reveals lower figures than any other condition, with the exception of meningitis.

"The micromethod for estimating the reducing substance of the spinal fluid has proved to be of value owing to its simplicity, reliability, and the small amount of fluid required.

"Fehling's test is unreliable and misleading unless the proper dilutions are constantly used together with equal parts of the fluid and the solution. In the latter case fairly constant relative results are derived when there is a marked change in the amount of glucose and when there is not an excess of protein. Quantitative estimations of the glucose concentration of the spinal fluid are of distinct value from the standpoint of very early diagnosis and prognosis, especially in meningitis."

Oscar M. Schloss and Louis C. Schraeder<sup>8</sup> agree that the reducing substance in the cerebrospinal fluid is a fermentable dextro-rotary sugar, probably dextrose. In infants and children the cerebrospinal fluid sugar ranges from 0.05 to 0.134 per cent. approximately the same as for blood sugar. There is no decrease in the reducing

(8) Amer. Jour. Dis. Child, January, 1916.

power of the cerebrospinal fluid in meningism. A large proportion of the cases of tuberculous meningitis show a decrease in sugar content of the cerebrospinal fluid at some stage.

Walter Kraus and G. G. Corneille<sup>9</sup> conclude that the change in the amount of sugar in purulent meningitis is of great value from a prognostic point of view, as a rise is favorable and a fall unfavorable.

**Circumscribed Purulent Leptomeningitis Due to Frontal Sinusitis.** Samuel Leopold<sup>1</sup> calls attention to the fact that circumscribed leptomeningitis of rhinogenous origin has been rarely noted by the neurologist, and only in the last decade or so by the rhinologist. The literature on intracranial, oculo-orbital and cerebral complications secondary to disease of the accessory sinuses of the nose has become so extensive that more recent writers have concerned themselves with complications arising from individual sinuses. Purulent leptomeningitis from frontal sinusitis may occur with or without brain abscess; it may be in association with pachymeningitis or with an extra-dural abscess.

The seat of the lesion is chiefly on the convexity of one or both frontal lobes, with varying involvement of other portions of the brain. The symptomatology is that of sinus disease with accompanying meningitis. Three forms may be distinguished: a sudden apoplectic, quickly terminating; a slow, insidious, protracted type; and an intermittent form, with a long interval before the meningitic attack.

He reports two cases from which, as well as from a study of the literature, it is shown that the symptoms are frequently preceded by weeks, or even months of nasal catarrh, with frontal headache; or they may occur after only a few days, following an attack of influenza. The edema and discoloration of the eyelid with tenderness over the orbit frequently precede the cerebral symptoms when disease of the wall of the sinus or orbit is present. The frontal headache is present in nearly all cases, though pain in the head is not limited to that region. The pulse

(9) Jour. Laborat. and Clin. Med., 1916, Vol. 1, No. 9, p. 685.

(1) Jour. Amer. Med. Ass'n., May 27, 1916.

and temperature are not characteristic. Rigidity of the neck and Kernig's sign, though noted in both Leopold's cases, are not frequent symptoms. Paralytic symptoms are noted usually in a later stage of the disease. Irritability and restlessness, alternating with clouding of the sensorium, are sometimes the only meningitic symptoms present; and death sometimes comes before the development of paralytic symptoms.

The pure meningitis following frontal sinus disease is less frequent than brain abscess, but much more frequent than thrombophlebitis. This is analogous to Kerner's statistics on otitic meningitis. In 140 cases analyzed by Gerber, there were sixty-five cases of brain abscess, to fifty-one of meningitis.

The explanation of this phenomenon depends on the route of infection, which may be direct, through the interstices or necrotic walls, or indirect, through the venae perforantes of the sinus and orbit, or through the lymphatics. Leopold quotes Gerber that the most frequent route in the production of leptomeningitis is through disease of the dura, following disease of the posterior wall of the sinus. Disease of the anterior wall, with thrombosis of the longitudinal sinus, may produce it indirectly; and disease of the inferior wall, with necrosis of the orbital roof and thrombophlebitis of the veins, may also indirectly produce leptomeningitis. It is possible that the lymph channels may play as important a part as the diseased walls and veins. Zwillinger, in some recent experiments on animals, has investigated the relation of the perimeningeal lymph spaces—the subdural and the subarachnoid—with the lymph vascular nests of the frontal sinus, and has shown that a direct connection exists. Logan Turner thinks that they play a subordinate rôle, for the operating table and necropsies have shown the infection spread as the result of caries of the contiguous bone.

There have been few cases reported in which operation led to recovery. Gerber found three out of fifty-one cases of meningitis, those of Herzfeld, Piff and Luc. To this Leopold adds the case of Spiller, Shields and Martin, reported in 1908. This case, not noted in the reports of

Gerber and Onodi, was the first case ending in recovery reported in America. The recovery in this case was supposed to be due to exposure of the diseased area to the air, but recent studies in experimental meningitis, by Kopetzky and Haynes, show that relief of intracranial pressure can bring about recovery.

It seems that in many cases the dura escapes involvement, and that incision is necessary in all cases in which absence of lesions on the surface is noted. This holds true, says Leopold, not only for the meningitic cases, but also for the subdural and frontal lobe abscesses.

The failure of recovery in most of these cases may be traced to several factors—the virulence of the infection, frequently streptococcus, with rapid termination of the case in several days; the absence of meningitic symptoms, the failure to recognize the underlying process sufficiently early and, finally, the presence of an apparently normal dura, preventive of further operation.

**Influenzal Meningitis with Report of a Case.** In 1911 Flexner published a summary of the information possessed regarding this condition up to that date, and earlier in the same year Wollstein reviewed this subject thoroughly, collecting forty-nine cases from which the influenza bacillus was recovered in pure culture and nine in which it occurred with other organisms. Simon, at about the same time, reported two cases and collected forty-one, including twelve not in Wollstein's series. These two series combined gave a total of sixty-one cases of pure infection and nine of mixed, with only five recoveries. Since that time Torrey has noted reports of twenty-six additional cases with two recoveries. A review of these reports tends to confirm previous figures regarding the high mortality of the disease. The cases reported by Robert G. Torrey<sup>2</sup> (including eighty-nine from the literature) show only eight recoveries.

Torrey's patient apparently had a severe attack of influenza with frontal sinus infection, followed by typical cerebrospinal meningitis. The organism isolated from the case showed a low degree of pathogenicity when tested by injection into rabbits. The spine was tapped thirteen

(2) Amer. Jour. Med. Sci., September, 1916.

times, and the influenzal organism was found present up to the last two tapplings. There was an improvement of the symptoms following each tapping, both when the serum was used and when it was not employed. The fluid was more turbid following the first injection of anti-meningococcus serum. It remained turbid as the organisms became fewer. The organisms at first were to a certain extent extracellular, but after the first injection of serum, they were almost always within the cells. The temperature was irregular throughout, but it was noted that following each tapping or injection there was a rise in temperature for a few hours. The use of the influenza serum was discontinued because the patient seemed to be improving, and because of the pain occasioned by its injection. With the clearing up of the organisms in the fluid, the temperature remained at normal, and the stiffness of the neck, which was the last remaining symptom, gradually disappeared.

Recovery was apparently complete with no nervous symptoms remaining. The only signs of high blood-pressure noted were extreme restlessness and irritability, which cleared up promptly with the removal of from 25 to 40 c.c. of fluid.

In another case reported, the patient lived for three days after admission to the hospital. He had frequent convulsions and attacks of vomiting and was stuporous throughout. The temperature varied from 102 to 105 F.

Torrey says that regarding treatment there are three measures recommended which deserve serious consideration.

1. The anti-influenzal serum of Wollstein, whose action appears to be specific. Where practical, this serum should certainly be tried.

2. Hexamethylenamine, which has been recommended by a number of writers, notably Brem and Zeiler, and by Batten. The latter reports a case in which recovery followed its use. There seems, however, to be less basis for regarding it as of great value than is the case with either Wollstein's serum or repeated spinal puncture and drainage.

3. Repeated lumbar puncture.

## CEREBROSPINAL MENINGITIS.

**Rashes in Cerebrospinal Meningitis.** Of considerable interest is the description of the various rashes given by C. A. Stewart.<sup>3</sup>

"The rash is a comparatively early symptom. Many of our cases have been admitted with the rash well formed, and in others it has appeared within a few hours of admission. There have been several forms of eruption, and the distribution is full of interest. The commonest form consists of small, petechial and purpuric spots, diffusely scattered over the trunk and extremities. The trunk appears to be the commonest site of eruption. The next situations in order of frequency are first the lower extremities and then upper extremities. I have only seen two cases in which there was any sign of a rash on the head. The cases were severe, and there was a single purpuric spot on the forehead and on the eyelid respectively. In severe cases, small hemorrhages may appear under the conjunctivae. The rash just described varies in intensity from one or two isolated spots to a most profuse eruption. When the eruption is sparse, I have found a very favorite site in the neighborhood of the hip joint. The rash may develop into hemorrhagic blisters.

"The second form of rash is decidedly interesting, both on account of its nature and distribution. It also appears early, and may or may not be associated with the first form. When it is so associated it is often not very marked until about twenty-four hours after the first rash has made its appearance.

"This rash has an appearance which I can only describe as 'hemorrhagic goose-skin.' The skin has the ordinary goose-skin appearance, but in addition, there is, first of all, a redness of the elevated papillae, and small hemorrhages occur into them after a short time. In many cases the blood actually finds its way out on to the surface of the skin, and on clotting forms many small scabs, several of which may coalesce to form a comparatively large one.

"The situation of this rash is practically constant. It

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(3) Med. Jour. of Austral., November, 1916.

is only seen on the extensor surfaces of the elbow and upper arm, on the skin over the great trochanters, and on the extensor aspects of the knees. The above order is also that of the frequency with which the eruption occurs.

"Associated at times with the 'hemorrhagic goose-skin,' but also occurring by itself, is a peculiar hyperemic flush in the same situations. This flush has at first sight all the characteristics of a pressure mark, but is differentiated from it by several facts. In the first place, it does not disappear on release of pressure; for instance, it is seen in an elbow after the patient has been lying on the opposite side for a considerable time. In the next place, it occurs round the knees and great trochanters, where there has been no pressure whatever.

"In the majority of the earlier cases this flush, which I have called the elbow or hip sign, was a very constant feature, and was often to be seen before the rash appeared. Latterly, it has been distinctly less frequent.

"The elbow is the commonest site of this flush, while the hip is the next. Even when there is a marked hip sign, it usually does not put in an appearance until a day or two after the elbow sign."

**Carriers in Cerebrospinal Fever.** It was found by H. Warren Crowe<sup>4</sup> that if contacts are segregated the proportion of carriers may rise: The percentage of positives found among twenty-one men examined on February 22 was 57.15, among sixteen men examined on March 2 it was 82.61, while on March 9 twenty men yielded 100 per cent. It is clearly then bad practice to isolate together all contacts of a case. Proof of the carrier condition should be sought at the earliest possible moment, and only those found positive segregated.

*Treatment of carriers.*—Broadly speaking, the greater the effort the smaller the success. Crowe tried a variety of methods and the net result seemed to be that the stronger the antiseptic employed the more obstinately did the meningococcus refuse to be eliminated. He was led to the conclusion that Nature assisted only by the weakest of washes—for example, permanganate of potash 1 in 10,000 or entirely unassisted, was more successful in

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(4) *Lancet*, Nov. 20, 1915.

removing the invader than the hand of man with his douches, swabs, or sprays containing antiseptic mixtures, however cunningly devised—a humiliating but, he believes, an incontrovertible truth.

In an abstract of the Reports of the Local Government Board on Public Health and Medical Subjects in Series 110, R. J. Reece<sup>5</sup> states that the corporation of Salisbury established under the direction of Dr. Penfold, of the Lister Institute, a bacteriologic laboratory for the diagnosis of cases and for the detection of carriers. The inhabitants were advised to submit to inoculation with a vaccine prepared from killed cultures of a local strain of the meningococcus, and the municipal health officer, Dr. Fison, estimates that at least 3,500 persons received preventive inoculation—that is to say, about 31 per cent. of the census population. Special attempts, however, were made to secure the inoculation of all contacts, and the results were such as to encourage resort to this measure in any future outbreak. Dr. Fison reported that the reaction from the inoculation was not severe, that no inoculated person contracted the disease; in two instances all the junior members of a family had been inoculated except the one attacked, and in another the father was attacked, but his inoculated children escaped.

**Treatment of Cerebrospinal Meningitis.** For the first time in the medical history of the British Isles, cerebrospinal fever has prevailed as an epidemic with the usual characteristics, *i. e.*, a low case-incidence, a widespread yet capricious distribution and predilection for soldiers, particularly young soldiers, a high case-mortality, and a rapid disappearance of the disease with the approach of fine weather. The figures of its distribution and prevalence are not yet available.

Sir William Osler<sup>6</sup> asks: "What preparations should be made in the prospect of such a renewal? Let us recognize a certain value in the conception of the disease propounded originally by Dopter, and supported in this country, among others, by Arkwright, Lundie, Thomas, Fleming, and Maclagan. These men hold that the epidemic is in the carrier; the meningitis is regarded by

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(5) Brit. Med. Jour., May 20, 1916.

(6) Practitioner, January, 1916.

them as an incident. The germ is ubiquitous, and is harbored by many who show no symptoms of the disease, but producing in others a mild catarrh, and only in a few reaching the meninges. That is what happened in the case of pneumonia, in which the proportion of cases to carriers is even smaller than in the case of cerebrospinal fever. The germ may be present in the throat without the occurrence of nasopharyngeal catarrh; it may be present in a severe case of the disease, without any indication other than that obtained by cultures.

"Two broad facts stand out: (1) the correlation of the seasonal prevalence with nasopharyngeal catarrh, and (2) the influence of overcrowding in ill-ventilated barracks and houses."

The concentration of human beings, particularly of young recruits in camps, seems to Osler to be the most important single factor. Fatigue lowers resistance; wet and cold and the excessive use of tobacco favor pharyngeal catarrh; too often the huts and tents are overcrowded, nights are cold, blankets thin; what wonder that the men close the windows, and lace up the tents tightly, so that the air becomes foul. The carrier does the rest, distributing the germ to a young fellow whose resistance is weakened, or whose nasopharynx forms a suitable medium.

On present knowledge, specific therapy combined with lumbar puncture is the rational treatment, combating the sepsis by means of one and the pressure effects of the local exudates by the other means. Physicians are agreed as to the value of withdrawal of fluid from the spinal meninges. On the value of specific therapy, however, there is a grave difference of opinion. The use of the serum is based on sound experimental data.

"We must be disappointed in many quarters with the serum treatment. The reasons for the failure should be laid open freely and frankly; what we wish is to get a statement of the truth of the matter. The consultant only sees severe cases, and cases which are far advanced. It has been said that when the cases reach us, they are in a condition to receive 'extreme unction.' The death-rate has been high, and I hope the figures for the whole country will be available.

"We want to know the reasons for this widespread failure. I think there has been one main cause: inert sera."

In trying to find an explanation of the increase of the death-rate from 24 to 36 per cent., A. Gardner Bobb considers:

1. "Regarding the virulence of the type, it is very difficult to make an estimate, but my opinion is that the recent outbreak was not more severe in type than formerly;

2. "The average age of the patients has been higher in the recent outbreak. This should, on the results generally reported in former outbreaks, make rather for a lower case-mortality, but my own experience has been that young children give very good results when treated with serum, provided they come under treatment reasonably early.

3. "Is the increase in the mortality to be accounted for by differences in the strain—strains not provided for in the polyvalent serum available? Having had some correspondence with Dr. Flexner on these resisting cases, I collected specimen cultures of the organism from cases in various places, and took them over to New York for comparison with the strains being used in the preparation of the serum. They showed no marked differences, when compared with those in use in the preparation of the serum now being made at the Rockefeller Institute.

4. "Was the serum available of as high a standard of immunity value. For two or three years before our epidemic last winter, there had been very little demand for the serum from the sources of supply in America; then suddenly came the great demand from this country. Our War Office ordered large quantities, and much was requisitioned from France, with the result that the available horses were bled as frequently as possible and the immunity value of the serum dropped very seriously. For this, and possibly for other reasons, I think there is no doubt that much of the serum which reached this country last winter was of a much lower standard of value than that formerly supplied. I think here lies the true reason of the somewhat disappointing results sometimes obtained from its use."

Alexander Presslie and W. E. Lindsay<sup>7</sup> report adversely on the use of serum in the treatment of cerebrospinal meningitis.

At first they used Mulford's, Burroughs Wellcome's, and Parke Davis's serums, but gave them up as they became quite convinced that they did no good, and for a long time treated the patients with simple lumbar puncture. Recently they have used the Lister Institute serum made for the War Office, but can not say that the results are any better than with simple lumbar puncture.

In February they had ten patients; eight recovered and were sent to the base; two died, making a death-rate of 20 per cent. In April they had also ten patients. One man recovered and was sent to the base; four are convalescent and are now awaiting evacuation; three have died; two are still seriously ill. The death-rate is then 30 per cent. up to the present. The February patients received no serum; the April ones all had serum.

The authors advocate daily lumbar puncturing, emptying the spinal canal on each occasion *as much as possible*. They have used this as the routine treatment and continue until all pressure symptoms are gone, such as headaches, and even then, if the temperature is still above normal, they continue it until the temperature has been normal for at least four successive days. They always withdraw fluid until it is below the normal pressure, and have seen no bad results, either temporary or permanent, from this. With very few exceptions the patients had a general anesthetic, chloroform being usually employed. They have found that more fluid can be withdrawn under a general anesthetic than if nothing is given. The daily dose of chloroform seems to do no harm, for the common rule is to give the patients chloroform ten or twelve times in successive days on an average, and no bad effects have followed.

H. A. Walsh and Wolfe S. Brown<sup>8</sup> sum up certain causes of failure in the treatment of cerebrospinal fever.

1. The most important is the failure to begin specific

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(7) Quart. Jour. Med., July, 1916.

(8) Med. Jour. Austral., August, 1916.

treatment at the onset of the infection, thus allowing the toxemia to develop.

2. Next is the failure to concentrate 60 c.c. of antiserum into the first twenty-four hours of treatment.

3. There is the failure to relieve subdural pressures when necessary—an indication often overlooked when fluid is poured out after injections of antiserum.

4. A possible cause of failure is excessive repetition of antiserum, which may be not only wasteful but hurtful. Apart from great virulence, delayed treatment, and relapses, it is seldom advisable to continue giving antiserum longer than they have recommended. The summation of reactions from a daily overdose of antiserum may result in an arrest of improvement, until the antiserum is cut off and lumbar puncture is done more frequently.

5. Some of their patients relapsed before the final recovery. In practically every instance the relapse was due to a failure to appreciate and carry out the later stages of intensive treatment. In every case the relapse was successfully met by massed doses of antiserum and more frequent punctures.

**Complement Re-inforcement of Antimeningococcus Serum in the Treatment of Cerebrospinal Meningitis.** As cerebrospinal fluid and antimeningococcus serum are both devoid of complement, and as the full effects of a bacteriotropic serum are not available in the absence of complement, R. J. Bull and N. Hamilton Fairley<sup>9</sup> recommend the complement re-inforcement of antimeningococcus serum in the treatment of this disease, as likely to lead to a further reduction of the mortality under serotherapy.

They state that the human serum from convalescent cases of cerebrospinal meningitis has invariably been used for complement re-inforcement. The amount was generally 5 c.c. human serum and 20 c.c. antimeningococcus serum. Since such small quantities of human serum were used, any increased value of re-inforced serum over that of simple antimeningococcus serum must be attributed rather to the addition of complement than of specific

(9) Med. Jour. Austral., February, 1916.

antibody. McKenzie and Martin's average injection was 15.20 c.c. of human serum in their reported cases; they used it to supply specific antibodies rather than complement.

## THE SYPHILITIC DISEASES OF THE NERVOUS SYSTEM.

**Pathognomonic Alterations of the Cerebrospinal Fluid in Syphilis of the Nervous System.** Joseph Collins<sup>1</sup> concludes that in cerebrospinal syphilis (lesion predominantly of the meninges and blood-vessels) the blood serum gives a positive reaction (if the patient has not been subjected to antisyphilitic treatment) in about 75 per cent. of the cases.

The cerebrospinal fluid, on the other hand, gives a positive Wassermann reaction in only about 30 per cent.

The most constant alteration is pleocytosis. It occurs in over 80 per cent. of the cases. In the majority of instances the cell count is less than 100 to the cubic millimeter, though it may be more than 2,000. Although treatment (salvarsan, mercury) usually reduces the number of cells, it does not always do so. Occasionally the pleocytosis increased within forty-eight hours after treatment. The predominating cell is the mononuclear, but the higher the cell count the greater the admixture of polynuclear elements.

The globulin content of the fluid is increased in about 45 per cent. of the cases.

The fluid of patients with cerebrospinal syphilis reduces Fehling's solution in about 95 per cent. of the cases, but this has practically no diagnostic value, as the fluid taken from patients who have any organic nervous disease does the same thing. When a fluid does not reduce Fehling's it will be found usually to contain a large proportion of polynuclear cells.

*Tubes:* Lymphocytosis is the most constant alteration of the spinal fluid (75 per cent.). The number of lymphocytes in some instances suddenly increases while the

(1) Amer. Jour. Med. Sci., February, 1916.

patient is under active treatment. The next most constant alteration is a + Wassermann (73 per cent.). The globulin is increased in 50 per cent. of the cases.

**General Paresis:** The cerebrospinal fluid gives a + reaction in from 80 to 85 per cent. of the cases; an increase of the cellular elements in practically the same percentage and globulin increase in about 65 per cent.

The colloidal gold test is positive in 97 per cent. of the cases.

**The Wassermann Reaction in Relatives of Syphilitics Especially Paretics.** Ernst Jolowicz<sup>2</sup> examined the serum of seventy-one individuals in thirty-three families. Of these twenty-nine were families related to paretics, one tabetic, two lues cerebri, and one latent lues. The original Wassermann test was used and seventy-one inactivated sera gave 21 per cent. positive reactions; fifty-one activated sera gave 54.9 per cent. positive; seventy-one active or inactivated gave 39.4 per cent. positive.

**Cultural Experiments with the Spirochaeta Pallida Derived from the Paretic Brain.** Udo J. Wile and Paul Henry De Kruij<sup>3</sup> describe the successful implantation of spirochetes and the transmission of experimental syphilis to rabbits from the living paretic brain. At present this experimental syphilis has been transmitted to five generations of rabbits.

The initial inoculation of brain was made into the testis of a large rabbit, June 11, 1915, and thirty-two days later, July 11, large numbers of spirochetes were demonstrable in both testes by aspiration. August 12, about two months after the initial inoculation, the right testis was castrated under aseptic precautions, one portion being used for implantation into other animals, and the remaining portion for cultural experiments.

At present, over four months have elapsed from the time of the initial inoculation, and the organism has been carried through three successful subcultures, at the end of which time these are as actively motile and as rich as at first.

The question of a neurotropic strain with certain morphologic characteristics, different from other spirochete

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(2) Neurol. Centralb., February, 1916.

(3) Jour. Amer. Med. Ass'n., Feb. 26, 1916.

strains, has been suggested by the work of Noguchi, Nichols and Hough, and others. Clinical evidence points suggestively to the existence of such a strain. In the organisms which the authors have isolated from general paresis, no morphologic difference is demonstrable. In the rabbit testis, numerous short thick forms with few spirals were found. In the cultures from this material, however, the predominating form was the typical fine spirochete with many convolutions, identical, in fact, with those seen in early cutaneous syphilids.

It is worthy of note, however, that although the authors had rich material to begin with, the cultures grew much more slowly and much less luxuriantly than do those cultivated from the early cutaneous or mucous membrane syphilids. On the other hand, it is to be noted that they are extremely viable.

#### **The Wassermann Reaction in Paretic Dementia.**

M. Edel and A. Piotrowski,<sup>4</sup> from an examination of cases of beginning paresis, conclude:

1. The history of lues is very often absent. The Wassermann reaction in the serum is regularly negative, somatic psychic symptoms fail entirely or are only slight, therefore the diagnosis is difficult.

2. When the Wassermann is positive in 0.1 c.c. of spinal fluid, it is a sure indication of the disease of the central nervous system.

3. The positive Wassermann reaction in the spinal fluid, and especially in small quantities of fluid, from 0.1 to 0.2 c.c., is the earliest sign of paresis and belongs to the pre paresis stage.

4. These cases speak against the statement that a negative Wassermann in the serum is a strong point against the assumption that paresis is present. It is far more correct to assume that the Wassermann reaction in the serum of early cases of paresis is negative.

Finally, the authors state that the only specific symptom of paresis in beginning of paresis, with a negative history and negative somatic psychic disturbance, is a positive Wassermann in 0.1 c.c. of the cerebrospinal fluid.

**Early Death from Cerebral Syphilis, with Successful Rabbit Inoculation.** The interesting case of a man

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(4) Neurol. Centralbl., Jan. 16, 1916.

whose death was due to cerebral syphilis, ten and one-half months from the date of infection, is reported by M. A. Reasoner.<sup>5</sup> He was treated adequately during the first three and one-half months, and received extensive mercurial treatment and two salvarsan injections during the last two months. He presented no signs of spinal irritation, special sense involvement, or other localizing manifestations.

The spinal fluid in this case contained the *Spirochaeta pallida* as was shown by injection into the testicles of a rabbit.

If there be, says Reasoner, such a thing as a highly invasive strain or one having a predilection for the nervous system, any characteristics peculiar to such a strain should be manifest in this one. Two other cases, in which infection was contracted from the same source, are now being followed. The results of the study of this strain in the rabbit will be published later in conjunction with the findings from a number of other strains, obtained from various sources. It is interesting to note that this strain shows the same tendency toward eye involvement in the rabbit, noted by Nichols, in the Nichols and Hough strain, obtained from the spinal fluid of a neurorecidiv.

**Treatment of Syphilis of the Central Nervous System.** Before discussing the treatment of syphilis of the central nervous system, Homer F. Swift,<sup>6</sup> at the Congress of American Physicians and Surgeons held in Washington, in May, 1916, made some remarks tending to help us to orient ourselves in the various problems which this disease presents. He said that it is now well established that spirochetes circulate in the blood of practically all patients in the late primary and early secondary stages, and during this period the central nervous system is frequently involved. In fact, with the early and almost universal dissemination of the infectious agent it is difficult to conceive how any organ escapes infection. It is not alone the presence of the spirochetes, but the reaction of the tissues toward them which determines the type of infection. It is recognized that spirochetes can lie dormant in the tissues, in latent cases, with practically no

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(5) Jour. Amer. Med. Ass'n., June 17, 1916.

(6) Amer. Jour. Med. Sci., 1916.

cellular reaction about them. Different observers report wide variation in the relative frequency with which the cerebrospinal fluid shows evidence of alteration in the secondary period. Ravaut found abnormalities, with pleocytosis or globulin excess, or both, in 67 per cent. of cases, Altmann and Dreyfus in 66 per cent. Gennerich states that at some time, either before or during intensive salvarsan treatment, 90 per cent. of his cases in the secondary period showed some abnormality of the cerebrospinal fluid. Ellis and the author found only 33 per cent. of abnormal fluids in untreated patients in the secondary period, but had the fluids in all patients in the secondary period been included in their statistics the percentage would be somewhat higher.

From the above observations it is evident that infection of the central nervous system occurs in a large proportion of cases of secondary syphilis.

It goes without saying that the best preventative of syphilis of the central nervous system is the prophylaxis of syphilis; but this problem is far from solved. Next in importance is the proper treatment of syphilis in the early stages. It should be emphasized at this point that no patient should be released from treatment until the cerebrospinal fluid has been shown to be normal, insofar as pleocytosis and Wassermann reaction are concerned.

After discussing the complications of the problem of treatment of the central nervous system induced by the peculiarities of the subarachnoid space, and the separateness of the subarachnoid space from the general lymphatic, and blood vascular systems, Swift states that the desirability of subarachnoid therapy has been well established. Reviewing the use of various agents and methods of their administration he remarks:

"Suffice it to say that the preparations which have stood the test of time are (1) the serum obtained from patients shortly after intravenous injections of salvarsan; (2) serum to which small quantities of salvarsan have been added; (3) neosalvarsan in small quantities and weak concentration, and (4) mercurialized serum."

The direct application of mercury in the form of albuminate, as devised by Byrnes, theoretically should be of value, Swift thinks, and the reports seem to indicate

that if the amount of mercury is kept under the irritating dose, beneficial results may be expected from its injection. He calls attention to the danger of repeated injections of mercurialized horse serum, which may lead not only to a general anaphylactic state of horse serum, but may cause the meninges to become hypersensitive to the foreign protein, and repeated injections over a number of months may lead to a chronic meningitis similar to the condition we are trying to combat.

The injection of neosalvarsan in concentrated solutions, as recommended by Ravaut, or even in one promille solutions has been proved by numerous observers to be a dangerous procedure, since it is apt to be followed by various degrees of urinary retention and incontinence, rectal paralysis, paresthesia of the legs, ataxia and girdle sensations, and, in severe cases, by paralysis of the lower extremities and death. A given dose may be well tolerated several times.

The treatment of small quantities of salvarsan to serum as recommended by Ogilvie was devised to give a known amount of salvarsan instead of the uncertain amounts in the serum salvarsanized *in vivo*. Marinesco and Minea have noted that neosalvarsan is less irritating when diluted with serum than when dissolved in water or saline. Both Ogilvie and Fordyce, however, have observed bladder disturbance and paresthesia in the legs when more than 1 mg. of salvarsan was added to the serum, and now Swift recommends that the dose should never be over 0.5 mg., usually less. With these quantities, intraspinal treatment has been followed by distinctly beneficial results.

The use of the serum of patients withdrawn after intravenous salvarsan injections has been criticized chiefly because of the small amounts of salvarsan it contained.

The autosalvarsanized serum is, however, definitely spirocheticidal and the salvarsan is present in a colloidal combination with the serum which cannot be dialyzed through a celloidin membrane, as can salvarsan alone (Young). Salvarsan in this colloidal state is probably more slowly diffused out of the cerebrospinal fluid, hence is longer in contact with the syphilitic exu-

dates. Moreover, it seems to Swift that some of the beneficial effect from serum injection may be due to the introduction of the serum *per se*, for he has obtained interesting results from the intraspinal injection of non-salvarsanized serum both in diminishing pleocytosis and strength of the Wassermann reaction.

A *résumé* of the results in twelve cases, treated only intraspinally with either normal or salvarsanized serum, shows that in half of them the reaction became negative with 1 c.c. of fluid, and in others there was distinct diminution of the reaction.

*Technique:* In most of Swift's work at the Rockefeller Hospital the blood was withdrawn within one hour after intravenous treatment and the serum diluted to 40 or 50 per cent. with normal saline and injected in quantities of from 30 to 40 c.c. of this dilution. It was thought that the dilute serum was less irritating, but for the past two years at the Presbyterian Hospital Swift and his colleagues have bled the patient one-half hour after intravenous treatment and injected 15 c.c. of whole heated serum without any more reaction than with the older method. Most of the patients now receive both the intravenous and intraspinal injections on the same day and return to their usual occupations the following day. In this way a minimum of time is required. It is necessary to keep some patients in bed for longer periods. In such instances an effort is made to give the treatments on Saturdays so that the patients may be in condition to work by Monday morning.

It has seemed to Swift that the treatments are better borne if not repeated oftener than once in two weeks. This is especially true in tabetics or patients with spinal syphilis. In paretics or patients with cerebral syphilis the intervals may be shorter. At times it seems wiser to lengthen the intervals.

One general principle which should always be considered is that in any patient who shows evidence of involvement of the cerebral meninges or brain, salvarsan treatment should be preceded by a short course of mercury to prevent the possible occurrence of a Herxheimer reaction in the region of vital nervous centers. If the presence of gummata is suspected a vigorous course of

potassium iodide is often followed by marked improvement. It has seemed to Swift that the response of patients in the tertiary state to salvarsan has been more marked and lasting if the courses of salvarsan have followed treatment with mercury and iodides. Gummatous exudates resolve under this preliminary treatment and the spirocheticidal effect of the salvarsan is more readily brought into play.

In the meningitis of the secondary period the response to alternate courses of salvarsan and mercury has been prompt and permanent in all of the author's cases. Gen-erich is of the opinion that all these cases respond more rapidly to combined intravenous and intraspinal therapy.

In the tertiary forms of the disease, the so-called interstitial forms, alternate courses of mercury and iodides, and of salvarsan are usually followed by decided improvement, both clinical and in the condition of the cerebrospinal fluid.

In *tabes dorsalis*, because of the apparent sensitiveness of many cases to mercury, it is better to start the treatment with small intravenous doses of salvarsan, gradually increasing and giving a treatment every week for a course of from six to eight injections. If at the end of this course the Wassermann reaction in the fluid is considerably weaker a course of mercury may be tried, followed by another course of salvarsan, alternate courses being given with periods of rest until the fluid is brought to normal.

In the presence of a rapidly advancing case of *tabes* or of optic atrophy, in which it is desirable to arrest the progress of the disease, in an important organ, the institution of combined intravenous and intraspinal therapy at the beginning should be seriously considered, as rapidity of cure is the end to be accomplished.

Compared with the results in other forms of lues of the central nervous system the treatment of paralytica dementia has been disappointing.

He concludes that in the treatment of syphilis of the central nervous system we should never forget the patient, and that he may be suffering from disease of other vital organs. If there is marked ataxia, Fraenkel's re-education movements are useful; if he is emaciated, attention

should be directed toward diet and general nutrition. Most patients with this form of disease show a certain amount of mental abnormality. Here psychotherapy is of value. In other words, treatment must be individualized, and while a general plan should be followed in the various forms of the disease, each case may demand a certain deviation to meet the peculiar conditions.

The objects of therapy are threefold: (1) the cure of disease; (2) the amelioration of symptoms; (3) the prolongation of life. With the possible exception of paresis all of these objects may be attained in most cases of syphilis of the central nervous system. To be satisfied with the attainment of the last two without attempting to attain the first is to fail to apply all the means at our disposal.

In an attempt to estimate the value of the Swift-Ellis method of treatment of syphilis of the central nervous system, I. C. Walker and D. A. Haller<sup>7</sup> report in detail on all the cases treated by the three methods, intravenous salvarsan alone, intraspinal salvarsanized serum alone, and both intravenous salvarsan and intraspinal salvarsanized serum, at the Peter Bent Brigham Hospital, since its opening three years ago, in order to compare the results following each method.

Seventy-five patients with central nervous syphilis were treated with 450 intraspinal injections of salvarsanized serum and with 350 intravenous injections of salvarsan. At first only salvarsan was used, and a few patients improved rapidly. However, in many cases little or no improvement followed from three to six injections, so these patients were then given intraspinal salvarsanized serum in conjunction with the salvarsan, and they improved rapidly under the combined treatment (Swift-Ellis method). As the intraspinal treatment seemed to re-inforce the salvarsan, the authors desired to determine what results would follow the intraspinal method alone. For this method they selected patients with a negative Wassermann reaction in the serum and with positive findings in the spinal fluid. The results from this method closely paralleled those from the double method. Therefore, the following rule has been adopted at this

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(7) *Archiv. Int. Med.*, September, 1916.

hospital: Patients are first treated with intravenous salvarsan. If satisfactory results do not follow three or four such treatments, they are then given intraspinal salvarsanized serum in conjunction with intravenous salvarsan. Those who had a negative reaction in the serum are given only intraspinal salvarsanized serum.

The first part of this paper consists of three protocols which are followed in each case by a discussion. The first protocol consists of cases in which the patients were treated with from three to six injections of salvarsan alone; the second protocol is the record of patients treated with three or four injections of intravenous salvarsanized and intraspinal salvarsanized serum together, and the third protocol shows the record of those treated with three or four intraspinal injections of salvarsanized serum alone. The purpose of this part of the paper is to compare the results following a similar number of treatments given by the three different methods. By this comparison the authors found that the double (Swift-Ellis) method gave more rapid and more satisfactory results than were given by the salvarsan alone and, furthermore, that the intraspinal salvarsanized serum alone gave rapid and satisfactory results. These results were evidenced by improvement in the patient's symptoms, by a drop in the cell count and by a decrease in positiveness of the Wassermann reaction in the spinal fluid.

The latter part of the paper deals with the total treatment given to each patient by the three methods. To thirteen patients were given five or more intravenous injections of salvarsan alone. Four of these patients, three with cerebrospinal syphilis and one with syphilitic meningitis, had very recent infections and were relieved of their symptoms. The spinal fluid cell count was reduced to normal in three cases, and the Wassermann reaction became negative with 1 c. c. in three cases. The remaining nine patients had older infections and they showed little or no improvement in the spinal fluid findings.

To thirty patients were given three or more double (Swift-Ellis) treatments, and marked improvement followed in each case. Thirteen patients who were previously incapacitated were restored to working capacity; of eighteen with ataxia, eight were relieved, five were

markedly improved and five more were much improved. Twenty-five patients showed improvement in the spinal fluid Wassermann reaction; in ten cases with amounts of from 0.3 c.c. to 0.5 c.c., and in five other cases the reaction became negative with 1 c.c. The spinal fluid cell count became five or less in twenty-five cases. In four cases a period of two years had elapsed since treatment without any return of symptoms, in three others a period of eighteen months and in eight others six months or more. Four patients have shown some form of relapse. One with tabes had a relapse in ataxia, two with general paresis of the insane had a temporary relapse in mentality, while one with cerebrospinal syphilis developed a weakness in the legs probably of cerebral origin.

Seventeen patients were treated intraspinally with salvarsanized serum alone, and all were markedly improved or relieved symptomatically. Nine who were previously unable to work were restored to working capacity. Of seven patients with ataxia, four previously unable to walk at all, became able to work, and in the other three, in whom the ataxia was not so marked, there is great improvement. The Wassermann reaction in the spinal fluid became negative with 1 c.c. or 2 c.c. in eight cases, and in five others it was improved from 0.3 c.c. to 0.5 c.c. The spinal fluid cell count became more normal in nearly every case. In two cases of syphilitic meningitis, the patients were relieved in every way. Seven patients have been observed twelve months since treatment was stopped, and four others for six months or more, and they show no return of the symptoms. One patient with cerebrospinal syphilis had a return of headache after eight months, and two with tabes had a slight relapse in ataxia, which rapidly cleared up immediately after following treatment.

In one patient a provocative Wassermann reaction occurred in the spinal fluid following the administration of salvarsan, and in two cases a provocative reaction occurred in the spinal fluid following intraspinal treatment. Two patients with repeatedly negative reactions in the serum while under intraspinal treatment developed a positive reaction in the serum about the time the spinal fluid reaction became negative with 1 c.c.

Improvement in symptoms following treatment seemed to parallel rather closely the drop in the cell count, and those patients with high cell count seemed to improve symptomatically more rapidly and the cell count dropped more rapidly than occurred in those cases with low cell count. The only physical sign which was changed in these cases was that a positive Romberg in one case became negative. More benefit seemed to follow moderate after-treatment reactions than when no reaction occurred. Severe reactions are undesirable and may be avoided by less frequent treatments. In this series of cases no fatal or disturbing results followed treatment.

In cases in this series the total number of cells in the spinal fluid did not vary as a rule. In a few cases of tabes, during severe crises of pain, and in cases immediately following too frequent and too large intraspinal injections of salvarsanized serum, the cell count temporarily increased. Since the cell count has become normal, the patients in nine cases have received lumbar puncture repeatedly for over a year, and four cases for over six months, and no variation outside of two or three cells was found in a single instance. Many other patients received lumbar puncture at longer intervals since treatment was stopped and no variation in the cell count was found.

The authors conclude, therefore, that patients with syphilitic meningitis and cerebrospinal syphilis may be relieved symptomatically by intravenous salvarsan; the spinal fluid Wassermann reaction may become negative with 1 c.c. and the cell count may become normal. Patients with long-standing cerebrospinal syphilis and tabes may be benefited symptomatically following salvarsan, but little or no change occurs in the spinal fluid findings.

Patients with recent and those with late syphilitic meningitis, cerebrospinal syphilis, tabes and general paresis of the insane are markedly improved following the combination of intravenous salvarsan and intraspinal salvarsanized serum (Swift-Ellis method), and those who fail to improve under salvarsan alone do improve both in symptoms and in spinal fluid findings following this double treatment.

The intraspinal salvarsanized serum greatly benefits

patients with central nervous system syphilis is shown by the fact that those with negative serum reactions and with positive spinal fluid findings are symptomatically relieved by this treatment. In many patients the spinal fluid Wassermann reactions becomes negative with 1 c.c., the cell count becomes normal and a negative (Noguchi) globulin test is obtained following sufficient treatment with salvarsanized serum intraspinally without other medication.

Richard Dexter and Clyde L. Cummer<sup>8</sup> report on fifty-nine intraspinal injections of autosalvarsanized serum in ten cases—two of cerebrospinal syphilis, one of early pareses, and seven of tabes. They conclude that the results in six of these ten cases has been a symptomatic improvement so emphatic that the patients' economic efficiency has been restored. They are able to work and to enjoy life to all intents and purposes as normal individuals.

They feel that the best results will be obtained in cerebrospinal lues, and in tabes of the early or moderately advanced types. In far-advanced tabes the results are in most cases dubious to say the least. Their experience with paresis is so limited that they do not care to make any generalization. It seems probable, however, that very little can be done to improve permanently a paresis once it is well established.

In concluding they state that they believe the Swift-Ellis method is safe when the original technique is followed to the letter. The claim of the originators that it is a valuable adjunct to the treatment of syphilitic involvement of the central nervous system is sustained, they think, so far as tabes dorsalis and cerebrospinal syphilis are concerned. It is a method which is not essential in all cases, but which applied carefully and controlled intelligently will bring about definite amelioration in symptoms and in laboratory signs where other accepted modes of attack have failed.

[A critical judgment as to the exact value of the procedure is impossible from such reports, inasmuch as the patients received not only intraspinal treatments but intravenous as well, and in many instances mercury was administered in addition.—Ed.]

(8) *Archiv. Int. Med.*, January, 1916.

**Treatment of Paresis and Tabes Dorsalis by Salvarsanized Serum.** Henry A. Cotton<sup>9</sup> concludes that in the use of salvarsanized serum we have an agent which does cause definite arrest in paresis, which arrest includes important clinical symptoms, physical signs and a corresponding change in the biologic reaction from positive to negative.

To be effective, treatment must be begun in the early stages as advanced stages show no favorable reaction to treatment. The length of time is not always an indication of the severity of the symptoms but the majority of cases can not be helped after two or three years have elapsed.

The treatment must be persistent and uninterrupted, the amount of dose and frequency of treatment being graded to the condition of the patient. Tabo paresis should be continuously treated usually with small doses and not oftener than every three weeks. The remissions caused by treatment can not be compared to spontaneous remissions, as in the former the percentage is 355 and in the latter four.

The changes in the cell count, globulin content, blood and spinal fluid, and Wassermann reaction are the direct results of treatment and not to be compared with the variations found in untreated cases of paresis. The efficiency of treatment depends not on the type or the method used but on the stage of the disease. Hence the necessity of early diagnosis in paresis and prompt treatment.

**The Newer Methods of Treatment of Dementia Paralytica.** Karl Hudovering<sup>1</sup> says that considering as the criterion of treatment only the questions as to which method offers the best clinical results irrespective of accidents or damage done, then tuberculin and nuclein treatment uncombined with mercury offers the minimal results. The best method is the thorough antiluetic treatment, then follows the nuclein combined with antiluetic. If, however, one considers the question of accidents and damage done, then the tuberculin combined

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(9) Amer. Jour. Insan., January, 1916.

(1) Neurol. Centralbl., January, 1916.

with mercury, and nuclein not combined with mercury, are more acceptable.

**Drainage of Cerebrospinal Fluid as a Factor in the Treatment of Nervous Syphilis.** S. F. Gilpin and Thomas B. Early<sup>2</sup> ask: Why, if either arsenic or mercury are present in the blood and produce good results in all other forms of syphilis, no such results are obtained in syphilis of the nerve tissue? The answer given is that after the administration of either drug by way of the mouth, skin or blood-vessels, neither drug was to be found in the cerebrospinal fluid. Why? Is it because the choroid plexus will not allow substances such as arsenic and mercury to pass, or is it because, if they do pass into the cerebrospinal fluid, it must be by osmosis, and this does not take place because the pressure of the cerebrospinal fluid equals the pressure of the blood in the venous sinuses and capillaries?

If the latter is the case why not saturate the patient's blood with either salvarsan or mercury, and at intervals drain off the cerebrospinal fluid by lumbar puncture, lower the pressure in the cerebrospinal sac, and thus cause the drugs to osmose through the capillaries?

In carrying out the treatment the authors tried to drain their patients once a week. They drained off as much cerebrospinal fluid as would flow; from 20 c.c. to as high as 40 c.c.

They report three cases and conclude:

"We realize that these three cases prove nothing. We know that the time of observation is too short to eliminate the improvement in a case of paresis which may be due only to a remission, but we see that from a clinical and laboratory point of view these three patients as well as others we have under treatment are improved, and we feel that we are justified in making this preliminary report in order to stimulate work in this field. We do not see any improvement in reflexes such as the knee-jerk and pupillary reactions. We see marked improvement in mental and physical health with lessening of the ataxic symptoms."

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(2) Jour. Amer. Med. Ass'n., Jan. 22, 1916.

**Intraspinal Administration of Mercurialized Serum.**

From a study of forty-five cases consisting of tabes general paresis, cerebrospinal syphilis, tabes paresis, clinical tabes (serologically negative) and syphilitic hemiplegia, E. L. Hunt<sup>3</sup> reaches the following conclusions:

1. The reaction from mercuric chloride or sublamin in no way differed from the reaction which followed the administration of salvarsan.

2. The sublamin did not seem to give the same reduction in the cell count or clinical improvement as did the mercuric chloride.

3. No ill results followed these injections. The three deaths could be easily accounted for, as the patients were practically moribund at the time of the treatment, and were given the injections as a last resort.

4. The patients with tabes and cerebrospinal syphilis were helped more than any others.

5. Such improvement as did occur was evident in the feelings of the patient, in the sphincter control, in the pains, in the gait, and in the serology. There was no improvement in the reflexes.

6. Such intraspinal treatment can be given only at intervals of two weeks, because a cell count does not fall sooner.

7. The reaction obtained was one in which the cell count was first greatly increased, and then diminished.

8. The cell count and the globulin yielded much sooner than did the Wassermann.

**The Intracranial Injection of Salvarsanized Serum.**

Drew M. Wardner,<sup>4</sup> owing his inspiration to the suggestion of Levaditi and Martel, describes the technique of his method for the subdural administration of salvarsanized serum (Swift-Ellis) and reports upon fourteen cases.

Five patients improved sufficiently to be able to go back to their work, and to date have remained well for from seven to eleven months. At the end of eleven months one had a bad relapse. He was immediately brought back to the hospital, and (Wardner thinks this a point much in favor of the efficacy of the treatment)

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(3) Jour. Amer. Med. Ass'n., Feb. 5, 1916.

(4) Amer. Jour. Insan., April, 1916.

responded immediately to additional applications of the serum. At present he is well, mentally and physically, and has parole of the grounds. Three other well-developed cases have improved sufficiently to be put upon parole of the grounds and are doing efficient work about the hospital. Three others have shown fairly marked physical and mental improvement, but can not yet be trusted at large. Two patients have died. In both of these autopsies were performed, and both showed, grossly and microscopically, changes suggestive of general paresis. The immediate cause of death was in one case bronchopneumonia, and in the other, in which there had been right-sided convulsions at intervals, there was found a large cyst underlying the left motor cortex. This last case had previously shown well-marked improvement and at the time of death the man was on parole. Two patients have shown no improvement.

Both mental and physical improvement has generally been observed after the second or third operation, and has followed so closely on the treatment as practically to preclude the idea of coincidence. The maximum amount of improvement has occurred as a rule after six or seven treatments.

That the operation is not dangerous is shown by the fact that 102 have been performed without untoward results in any case. Thirty minutes suffices for the whole procedure, and the anesthetic has been well borne in all Wardner's cases.

The cases treated were not selected but were taken at random from the admissions of the past two years. Every attempt has been made to exclude psychoses other than general paresis, and in every case the clinical diagnoses has been supported by the laboratory findings.

Where pupillary disturbances existed no marked change has been noted after treatment. Reflexes, when previously exaggerated, show a tendency to become less so. Coördination and speech have been improved in all.

In the blood, the Wassermann reaction has been rendered negative in six, reduced in intensity in four, unchanged in three, and not taken in one. In the spinal fluid the Wassermann has been rendered negative in two, reduced in six, not retaken in two, unchanged in the

rest. The cell count has been reduced to below 10 per cm. in twelve; not retaken in two. The Ross-Jones reaction for globulin has been made negative in one; unchanged in the remainder.

What the final outcome of these cases will be Wardner can not, of course, state. His best results have occurred in those in which the manifestation of the disease process had been noticed within a comparatively short time, and in which the actual destruction of brain tissue might reasonably be supposed to be slight. He believes that if cases of general paresis could be diagnosed early and thoroughly treated much might be done to control the future progress of the condition.

Evans and Thorne<sup>5</sup> from their experience come to the conclusion that the intracranial methods seems unnecessary, and that the same results can be obtained with the intraspinal method.

[Intraspinal, intraventricular, and subdural methods of administering spirocheticidal drugs is of course based on such experimental work as shows that the meninges are impermeable to such drugs and that they are filtered out by the action of the chorioid plexus.<sup>6</sup> It must be remembered, however, that in such experimental work as resulted from the intravenous injection of dye stuffs as trypan blue, methylene blue, etc., that not only was the brain unstained but also the suprarenal glands, the testes ovaries and other glands; in other words the failure of these tissues in common with the brain was due to the fact that the stain employed was not organotropic for these tissues. It must also be pointed out that methylene blue when injected into the living body is reduced in presence of the alkaline tissues to its colorless leuco-base and therefore can not be demonstrated until it is oxidized and then if not properly fixed it rapidly fades. Certainly not all the reason for failures in the treatment of syphilis of the nervous system contributed to the impermeability of the conducting tissue. A factor inherent in the drug itself must be present.—Ed.]

**Tabes Dorsalis.** Baldwin Lucke<sup>7</sup> analyzes the symp-

(5) Amer. Jour. Insan., April, 1916.

(6) This was described in the Practical Medicine Series, 1914, Vol. 10, p. 30.

(7) Jour. Nerv. and Ment. Dis., May, 1916.

toms and pathologic findings in 250 cases of tabes dorsalis, and compares the results with similar statistics.

"Two hundred and seven or 82.8 per cent. are white males. Twenty-nine or 11.6 per cent. are white females. Thirteen or 5.2 per cent. are black males. One or 0.4 per cent. is a negress. The ratio between males and females is therefore 8.5:1.0.

Of Lucke's cases only 141 or 56.4 per cent. admitted lues. In forty-six cases it was noted whether secondary eruptions followed the chancre. Twenty-eight patients or 60.8 per cent. disclaimed secondary lesions.

In this series there were thirteen negroes and one negress; whether these are full-blooded Africans or whether admixture of Caucasian blood existed in some or all Lucke is unable to state.

It was found that 15.34 years was the average period which elapsed between primary sore and beginning tabetic symptoms.

The incubation period ranges from three to forty-five years (1 case) with the maximum percentage between 10-14 years.

The average age at which subjective symptoms made their appearance is difficult to determine, since patients will only seek treatment when their symptoms are troublesome; there is no age, however, in which tabes may not occur. In Lucke's cases it varied from 23 to 65 years. With the average of 42.34 years, the highest percentage of cases occurred between 35 and 39 years.

#### TABETIC SYMPTOMS AND SIGNS IN ORDER OF THEIR FREQUENCY.

	Per Cent.
1. Romberg sign.....	96.4
2. Absent knee-jerks .....	90.0
3. Lancinating pains.....	88.4
4. Staggering gait.....	87.2
5. Argyll-Robertson pupil.....	80.0
6. Ataxia in upper extremities.....	68.2
7. Sphincter disturbances.....	67.6
8. Sensory disturbances.....	58.2
9. Visual disturbances.....	43.6
10. Paresthesia and numbness of feet and lower extremities	42.8
11. Girdle sense.....	31.2
12. Ptosis of eye-lids.....	23.2
13. Paresthesia or numbness in hands or upper extremities	13.6
14. Strabismus .....	12.0

15. Visceral crises .....	12.0
16. Loss of sexual desire.....	11.5
17. Charcot joints .....	9.2
18. Vertigo .....	4.0
19. Mal perforans.....	3.2
20. Pain in joints.....	2.8
21. Rectal tenesmus .....	2.8
22. Mental degeneration (other than paresis).....	2.4
23. Hemiplegia .....	2.4
24. Vesical tenesmus .....	2.0
25. Difficulty in articulation.....	2.0
26. Deafness .....	1.2
27. Anosmia .....	0.8

## OBJECTIVE SYMPTOMS.

Visual disturbances occurred in	109 cases or 43.6 per cent. of these
Failing eye-sight occurred in	63 cases or 25.2 per cent.
Diplopia occurred in	46 cases or 18.4 per cent.
Optic atrophy occurred in	37 cases or 16.0 per cent.
Nystagmus occurred in	23 cases or 9.2 per cent.

## PUPILS, PTOSIS, AND PARESIS OF EYE-MUSCLES.

Typical Argyll-Robertson pupil	
occurred in	200 cases or 80 per cent.
Sluggish reactions or no reactions	
occurred in	32 cases or 12.8 per cent.
Normal reactions occurred in	18 cases or 7.2 per cent.
Unequal pupils occurred in	86 cases or 33.6 per cent.
Irregular pupils occurred in	34 cases or 13.6 per cent.
Unequal and irregular pupils occurred in	20 cases or 8 per cent.
Ptoxis of both eye-lids occurred in	17 cases or 6.8 per cent.
Ptoxis of left eye-lid occurred in	16 cases or 6.4 per cent.
Ptoxis of right eye-lid occurred in	4 cases or 1.6 per cent.
Ptoxis therefore occurred in	37 cases or 14.8 per cent.
Paresis of eye-muscles occurred in	30 cases or 12.0 per cent.

*Reflexes:* Only the patellar reflex is considered in this paper. It was found to be:

Absent on both sides in	217 cases or 86.8 per cent.
Absent on one side in	8 cases or 3.2 per cent.
Diminished in	11 cases or 4.4 per cent.
Normal in	6 cases or 2.4 per cent.
Increased in	8 cases or 3.2 per cent.

*Romberg's sign:* Romberg's phenomenon was present in 241 cases or 96.5 per cent. Nine cases or 4.5 per cent. showed no swaying standing with the feet together and eyes closed. Romberg's sign was found present by von

Sarbó in 93 per cent.; Limbach in 88.75 per cent.; Bonar in 79 per cent.; Thomas in 76 per cent., while Frey noted it in but 54 per cent. In women Mendel and Tobias found it in 81.7 per cent; Friedrichsen in 90 per cent. and Fehre in 71 per cent.

**Needless Surgical Operations from Failure to Recognize Tabes.** In the past five years (1910-1915) over 1,000 cases of tabes dorsalis have been carefully studied in the Cook County Hospital. These records have been examined by John W. Nuzum<sup>8</sup> to determine among other things the number of patients operated upon. Of 1,000 patients with tabes only five had operation in the Cook County Hospital during the past five years. Among those operated on, in ten different hospitals, there were twenty operations on tabetics under mistaken diagnosis in approximately the past four years.

The following are what have been apparently surgical errors:

Ninety-seven operations have been performed on eighty-seven patients. Of the total number, there were nineteen operations on eighteen patients for gastric ulcer; nineteen operations on sixteen patients for gall-stones or cholecystitis; eighteen operations on seventeen patients for appendicitis; eleven patients were operated on thirteen times for salpingitis; on nine patients an exploratory laparotomy was made; seven operations were performed on six patients for renal calculi; seven operations on five patients for post-operative adhesions, and one operation for each of the following: Tumor of the cauda equina, sciatica (nerve stretching), meningocele, ectopic gestation and peritonitis.

Of 1,000 tabetics, 8.7 per cent. have been subjected to laparotomy under mistaken diagnosis one or more times. Furthermore, gastric ulcer was the diagnosis most frequently made, and next gall-bladder disease, with appendicitis a close third. As regards operations for post-operative adhesions, there were seven on five patients. One patient had five laparotomies, the last three for post-operative adhesions, and following each operation the old symptoms of epigastric pain with severe vomiting returned, that is, gastric crises of tabes.

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(8) Jour. Amer. Med. Ass'n., Feb. 12, 1916.

From a study of the information presented in these charts, it is apparent that the mistaken diagnosis and resulting operations were chiefly in two main groups of patients:

1. Tabetics in whom the initial symptoms were the visceral crises and less frequently those with renal or intestinal crises.

2. Tabetics in the class designated by Erb as "*tabes incompleta*" or "*formes frustes*" by the French. These patients presented fewer of the characteristic symptoms of tabes, and the mistakes leading to operations are less surprising.

Nuzum's conclusions are as follows:

1. Of 1,000 tabetics, 8.7 per cent. have been subjected to laparotomy under mistaken diagnosis one or more times.

2. The "crises" of tabes have largely influenced the surgeon in his decision to operate. This statement is supported by the fact that 65 per cent. of the eighty-seven patients operated on presented visceral crises. In 17 per cent. of these, the "crises" were the *initial symptoms* of their disease.

3. Mistaken diagnosis and resulting operations occur chiefly through failure to examine the nervous system.

4. Gastric ulcer, gall-bladder disease and appendicitis are the diagnoses most frequently made.

5. Tabetics subjected to several successive laparotomies have, as a rule, been operated on by as many different surgeons.

6. A history of paroxysmal attacks of vomiting, rheumatism, paresthesias, bladder disturbances or fractures without physical violence should excite interest to exclude tabes dorsalis.

7. The cytodagnosis of the cerebrospinal fluid, together with the Wassermann reaction with the spinal fluid, are of intestimable value in doubtful cases.

**Tabetic Ocular Crises.** William G. Spiller<sup>9</sup> reports the following case:

A colored man, who has been under his observation many years at irregular periods and is now in the service of Dr. C. K. Mills, has ocular crises of tabes with

(9) Jour. Amer. Med. Ass'n., March 18, 1916.

phenomena of irritation of the visual fibers not previously observed in other cases. This man was presented before the Philadelphia Neurological Society in 1910 by Dr. F. X. Dercum, to demonstrate that closure of the eyes greatly increased his ataxia, although the man was blind.

The case is typically one of tabes in a colored person. He has difficult micturition, optic nerve atrophy, ataxic gait, Romberg's sign, shooting pain in the lower limbs, loss of tendon reflexes, etc.

The attack of pain in the eyes and adjoining parts date from 1901, and occurred with tabetic pain elsewhere in the body. During the attacks of ocular pain, peculiar visual phenomena developed. He described them as flashes of light, twisting and assuming the shape of reptiles, birds, houses, men, various forms of animals, etc. He asserted that he saw these objects clearly.

At the present time he describes the visual phenomena as follows: The attacks of ocular crises occur at irregular periods, and sometimes he has repeated attacks in a day; but the visual phenomena never occur without pain in the eyeballs. The pain is intense, jerking, stinging and burning, and the eyeballs feel as though they would come out of the orbits. The pain may last half an hour or longer in the eyeballs, and is accompanied by a free secretion of tears. The pain may suddenly remit and suddenly begin again with great severity. In the ocular crises he may see a body of water, as a pond, which revolves then bursts, and gives place to figures of live lobsters, crabs and fish, moving rapidly backward and forward, some being red and some brown. At times he sees beds of "magnificent" flowers of various colors, which would give him great delight if the pain in the eyes were not so intense. He sees also faces of human beings, but not of persons known by him, he also sees blue stars on a brown background.

This man has no mental symptoms, he is entirely rational and realizes that these visual phenomena have no real existence. He is totally blind and can not even recognize daylight.

Commenting upon the case Spiller says that it resembles the three cases of tabetic ocular crises reported in

the literature (Pel, Haskovec, Knauer) in the sudden onset of attacks of severe orbital pain, with free lachrymation, but differs in the occurrence of visual phenomena. The latter phenomena recall to mind the scintillating scotomas and visual disturbances of certain cases of migraine or epilepsy, but they can not be considered as identical with these. There is no evidence of cortical irritation in the ocular crises of this case. The lachrymation and pain probably are the result of irritation of the trigeminal nerves, and there appears to be a simultaneous irritation of the optic fibers resulting in visual phenomena. Spiller has known visual hallucinations to be caused by hemorrhage into the retina and has referred to a number of cases in which these phenomena were produced by irritation of different portions of the visual system.

## DISEASES OF THE BRAIN.

### VASCULAR DISEASES.

#### **Prodromal Symptoms of Cerebral Hemorrhage.**

Kisch<sup>1</sup> calls attention to the enlargement of the area of dulness over the heart and signs of sclerosis of the cerebral vessels and also of the vessels of the kidneys as signs suggesting impending cerebral hemorrhage in those inclined to corpulence and past 50, especially when associated with functional bowel disturbance. Long before the arteries feel hard, or show a tortuous course, a permanently high pulse-pressure reveals the tendency to arteriosclerosis with resulting overwork on the part of the heart. He found the heart hypertrophied in five of thirteen cases of fatal cerebral hemorrhage, Lowenfeld in twenty-seven of sixty, and Kirkes in seventeen of twenty-two. The signs of arteriosclerosis of the capillaries in the brain are frequent headaches, frequent transient vertigo, permanently restless sleep, forgetfulness, especially of names and figures, slight motor disturbances in hand or foot, sluggishness in moving the tongue, transient disturbances in speech, various sensory disturb-

(1) Med. Klinik, Feb. 27, 1916.

ances, chilliness in hands or feet, feeling that they "have gone to sleep," anesthetics and paresthesias. Among the latter he has noticed particularly a sensation of heat on the top of the head, or a burning patch on the skin from the size of a quarter to that of the palm of one's hand. Windscheid and Weber have recently described some mental symptoms and moods as suggestive of arteriosclerosis of the brain.

Kisch regards albuminuria as revealing arteriosclerosis of the kidney vessels in these patients. He has long warned that not enough attention is paid to albuminuria in the corpulent and obese. He found stasis hyperemia in the kidney in five of eighteen cadavers showing extreme obesity, parenchymatous degeneration in eight, and granular atrophy in two. Monakow found kidney disease in 30 per cent. of his apoplexy cases. The arteriosclerosis of the viscera is liable to become manifest in meteorism after each meal, a feeling of oppression and pain in the abdomen. Relief is obtained only by loosening everything tight around the body and resting for a time. Constipation and hemorrhoids are also common, the whole suggesting possibly what Ortnier calls intermittent intestinal angiosclerotic dyspragia; the pushing up of the diaphragm may add shortness of breath to the clinical picture. Hereditary influences are marked, the apoplexy coming on at about the same age in the different members of a predisposed family; a man and his four sons in one family all succumbed at the same age to apoplexy. Among the factors liable to bring on the catastrophe, besides over-exertion, over-eating, straining at stool and violent emotions, he lists sudden joy as well as anything that upsets the physiologic or psychologic balance, and sudden extreme vicissitudes of the weather.

**Aneurysm of the Internal Carotid Artery.** George J. Heuer and Walter E. Dandy<sup>2</sup> in reporting a case of bilateral aneurysm of the internal carotid artery comment as follows:

"The sudden onset with terrific headache and vomiting, followed by a long interval (four years) without marked increase in the symptoms, indicated a vascular lesion; although a sudden hemorrhage into a vascular

(2) Bull. Johns Hopkins Hosp., August, 1916.

tumor might have given identical symptoms. The *x*-ray picture was striking; although we had never seen a similar picture, in retrospect, we should have considered that in no condition would concentric layers of calcification be so likely to occur as in a thick-walled, aneurysmal sac. Although on the left side the wall of the aneurysm had eroded the orbit and lay against the fat behind the eyeball, there was absolutely no pulsation of the globe, that is, no pulsating exophthalmos, and no bruit on auscultation over the skull. This may be explained by the rigidity of the walls of the aneurysm due to the calcification and the almost complete filling of the sac with organized tissue. The cause of the sudden increased intracranial pressure leading to death was not determined; there was no rupture of either aneurysm (see Plate I.).

"The *x*-ray as an aid in diagnosis in intracranial conditions still finds its greatest field of usefulness in the diagnosis of hypophyseal lesions; not only in their localization, but, from the associated deformities of the sella turcica, in the determination of their size, direction of growth, and, occasionally, of their character. Aside from hypophyseal tumors, however, the study of the *x*-ray plates in this series has proved of great aid in the diagnosis of intracranial new growths; for, while tumors producing a definite shadow are unfortunately uncommon, changes in the skull, such as erosion or thickening, vascular changes or changes more or less characteristic of internal hydrocephalus, are not frequent. The value of the various phases of *x*-ray changes in intracranial lesions is considered in a paper which will appear shortly."<sup>3</sup>

**Bulbar Paralysis and Bilateral Exophthalmos from Cranial Nerve Involvement in Sinus Thrombosis Secondary to Otitis Media.** A. Halipre and Paul Petit<sup>4</sup> discuss the rare phenomenon of bulbar paralysis produced by affection of the cranial nerves outside the bulb, and report a case in a child. First there was bilateral otitis media with sinus thrombosis causing bilateral facial paralysis from compression of the facial nerves. Then followed thrombosis of the left superior and inferior

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(3) Cerebral aneurysms are reviewed in the Practical Medicine Series, 1914, Vol. X, p. 56.

(4) Rev. Neurol., October, 1915.

petrosal sinuses, of the right lateral sinus and jugular vein, finally of the ophthalmic veins with the development of the exophthalmos. Typical glosso-labio-laryngeal paralysis was present. A careful post-mortem examination was made.

#### MISCELLANEOUS BRAIN DISORDERS.

**Hydromyelia and Hydroencephalia.**—Alfred Gordon<sup>5</sup> reports a case in which the patient, a girl, until the age of 10 was in good health. Two years following a severe trauma a few symptoms developed. At no time did she present any mental disturbances and repeated examinations failed to reveal any sensory disturbances characteristic of syringomyelia. Also in spite of the bulb being involved no symptoms referable to that portion of the brain-stem were present. Pathologically Gordon found, besides the enormous dilatation of the central canal of the cord and of the ventricles of the brain with extraordinary deformity of nervous tissue, also marked vascular changes, *viz.*, thrombosis of the anterior spinal artery and of numerous small vessels within the cord and medulla. The latter were seen mostly near the anterior portion of the cord, *viz.*, near the thrombotic anterior spinal artery. Moreover a certain degree of meningitis was also present in the vicinity of the peripheral thrombotic arteries. The simultaneous occurrence of a marked dilatation of the cerebral cavities, also of the central canal of the cord, and the beginning of somatic disturbances at an early age together with almost total absence of the corpus callosum—all these facts speak in favor of a congenital malformation of the cerebrospinal axis. The vascular disturbances mentioned probably participated to a certain extent in the pathologic process of the cord but undoubtedly the malformation of the central canal, together with the presence of a considerable amount of gliomatous tissue, could not have been produced exclusively by a vascular lesion of that character, neither by the accompanying slight meningeal inflammation. Besides, the co-existing enormous dilatation of the brain cavities and absence of the corpus callosum were not

(5) Jour. Nerv. and Ment. Dis., May, 1916.

accompanied by conspicuous lesions of the cerebral vascular system (see Plate II.).

**Crossed Aphasia.** In referring to a condition which he described in 1899 as crossed aphasia, Byron Bramwell<sup>6</sup> says that in right-handed persons the "leading" or "driving" speech centers are situated in the left hemisphere of the brain, and *vice versa*, in left-handed persons (but probably less constantly) in the right hemisphere. Consequently, when marked and persistent aphasia occurs in a left-handed person, the lesion is situated in the right hemisphere, and the hemiplegia is left-sided, and *vice versa*. But in rare and very exceptional cases an attack of *right-sided hemiplegia in a left-handed person is attended with aphasia*. In other words, in exceptional cases, the "leading" or "driving" speech centers are in left-handed persons situated in the left hemisphere of the brain.

In giving the explanation of this condition he says that it is essential to remember the following facts:

1. The majority of mankind use one hand more than the other, *i. e.*, are either right-handed or left-handed.
2. The majority of persons are right-handed.
3. The ancestors of the majority of persons have for generations been right-handed.

Consequently, (a) heredity, (b) congenital organic aptitude, and (c) actual acquirement (habit, example, social usage and education) all tend to make the majority of persons right-handed.

It therefore follows that in right-handed persons the left hemisphere, and in left-handed persons the right hemisphere, is the "leading" or "driving" side (hemisphere) of the brain.

4. With rare exceptions the "leading" or "driving" speech centers are situated in the hemisphere of the brain, which is, so far as the movements of the hand are concerned, the "leading" or "driving" side.

He is disposed to think that some of the exceptional cases in which in left-handed persons whose forefathers were not left-handed, *i. e.*, who did not inherit left-handedness, the "leading" or "driving" speech centers are situated, not as one would expect in the right, but in the

(6) Edinburgh Med. Jour., May, 1916.

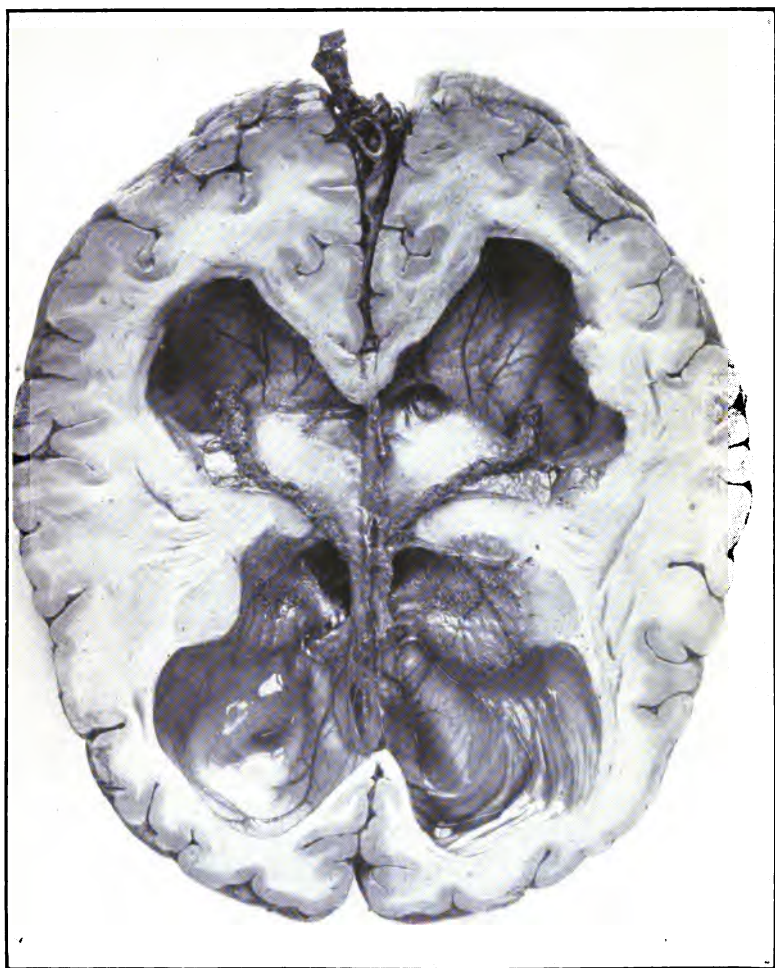
PLATE I.



Case of bilateral aneurism of the internal carotid arteries. The smaller aneurism upon the right side is covered by the larger mass.  
—Heuer and Dandy (see page 77).



PLATE II.



Hydroencephalia.—Gordon (see page 79).



left hemisphere of the brain, may perhaps be best explained by supposing that in them the influence either of (a) heredity, or (b) of heredity + the third factor which he has suggested (*viz.*, the learning to write and the practice of the act of writing with the right hand) was, so far as the localization of the "leading" or "driving" speech centers is concerned, stronger than the influence of the (non-hereditary) left-handedness; that is to say, in such exceptional cases the hereditary localization of the "leading" or "driving" speech centers in the left hemisphere (or heredity so far the speech centers are concerned + the third factor) have prevented the transference of these (the "leading" or "driving") speech centers to the right hemisphere.

Dr. Foster Kennedy<sup>7</sup> says that the cause of the indecision in regard to the location of the speech centers, and therefore, the causation of aphasia, lies in the fact that apparently irrefragable evidence can be produced by the protagonists of each of the different views. The case used to disprove the validity of Broca's are only impugns the theory of constant conjunction of right-brainedness and left-handedness or *vice versa*. He wants to suggest that the simple statement that a given patient is right-handed or left-handed is not adequate. After investigating the question of a patient's handedness only by investigating also the prevalence of the type of handedness in his stock shall we be able to throw some light on a very obscure chapter of neurologic medicine. In the literature of aphasia one finds that but few cases disagree with the general hypothesis that in right-handed persons the centers of language are situated on the left side of the brain. In the few instances in which aphasia has resulted from injury to the right brain, some anomalous and adventitious circumstance has usually been brought forward to account for the situation. It would appear that this trend when present in the stock may produce in a few right-handed individuals of the sinistral stock a condition of brain similar to their collateral relatives and ancestors, with the result that the speech area in such persons becomes developed in an ectopic position.

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(7) Jour. Amer. Med. Ass'n., June 10, 1916.

**The Phenomenon of "Tonic Innervation" and Its Relation to Motor Apraxia.** Kinnier Wilson and Walshe<sup>8</sup> understand tonic innervation or preservation to signify inability, owing to a central lesion, to relax a given innervation in any muscular group or groups; *e. g.*, a patient with the left hand affected, on grasping an object, was unable to let go. They record three cases, in two of which tumors were found in the precentral region, involving the upper surface of the hemisphere. The volitional element was the disturbing element, and the symptom was aggravated by voluntary alteration; it was a psycho-motor defect. The corticospinal system for the limb was not functionally normal, but at the same time not greatly involved. The phenomenon was explained by a disturbance of reciprocal innervation in "willed" movements. "As the patient voluntarily contracts his flexors, active inhibition of the antagonists, *viz.*, the extensors take place; when next he would reverse the action, he is able voluntarily to innervate the extensors, but there is no corresponding inhibition of the flexors." This constituted the symptom.

**The Possibility of Recovery of Motor Function in Long Standing Hemiplegia.** S. I. Franz, M. E. Sheetz, and A. Wilson<sup>9</sup> report the results of treatment in five long standing hemiplegias.

The results which they have obtained with five patients show very clearly the possibility of a return of function in a paralyzed segment even after the paralysis has existed much beyond the time limit set by some neurologists for possible improvement.

They state that so far as the results from these cases go they have the right to say that the time limit beyond which improvement may not take place can not be set. Furthermore, there appears at present no indication of a limitation of the quantity of improvement which may be attained by suitable and sufficiently prolonged therapeutic measures. It is also to be noted that these five cases differ with respect to the locations of the cerebral lesions (so far as these can be determined by clinical as contrasted with post-mortem examination). Case 1 is

(8) Brain, Vol. 37, Part II, 1915.

(9) Jour. Amer. Med. Ass'n., Dec. 18, 1915.

doubtless a purely cortical case; another (Case 5) has been diagnosed as cortical, and the other three have signs which indicate more plainly capsular lesions. Examination of the records shows that improvement has taken place almost equally well whether the lesion be cortical or capsular.

The underlying causes of the cerebral lesions differ. Case 1 is definitely traumatic; Case 5 is probably arteriosclerotic; Case 4 is definitely syphilitic; Case 2 is probably syphilitic, and Case 3 is only possibly syphilitic. The most marked and quickest improvement took place in Case 5, and the least in Cases 1 and 4. The authors do not consider that these facts reveal any relation of special disease to the return of function, both because they have too few cases of any one kind for a definite conclusion and because they find great individual differences in coöperation of the patients, which latter fact appears to be more important. Since the diseased conditions which they mention, arteriosclerosis, syphilis and traumatism, are the most frequent causes of hemiplegias and other cerebral palsies, it would appear likely that most cases of cerebral paralysis can be treated so that improvement will take place.

Moreover, the patients with whom they have dealt are patients who have been committed to an institution for the insane. They are, therefore, not mentally normal, and the authors could not expect and, in fact, did not get the amount of coöperation from them which can rightly be expected from mentally normal patients. Their experience with the rapid improvement in Case 5 leads them to the belief that improvement may be rapid when the active coöperation of the patient can be obtained, but that without this coöperation, or with a minimum of it, improvement may be produced but slowly and to a slight degree. This is shown by the slowness of the patient in Case 3, who was lazy and non-coöperative.

The authors think that the facts in these cases justify the conclusion that patients with any kind of cerebral paralysis should not be immediately consigned to the chronic, or incurable, or irrecoverable class, but should be intelligently dealt with just as any surgical case, and the effort should be made to relieve if not to cure. Every

effort should be made to put into use with these patients all the means at command to bring about a restoration of function or at least an amelioration of their helplessness. There appears to be no good reason why the discomfort and the deformity of the contractures in such cases should be permitted to exist.

Because of its popular pseudo-therapeutic connotation they hesitate to use the term "mechanotherapy," but urge that properly used massage, passive movements, hot baths and packs, followed or accompanied by attempting to obtain on the part of the patient voluntary muscular contractions, be employed in all such cases. They recognize that treatment of this character can not be given by any one, for, to speak only of massage, the ordinary slapping and rubbing which a patient may get at the hands of some well-meaning (but in this respect ignorant) relative or nurse may do more harm than good because the flexors may be stimulated much more than the extensors and the flexor condition be exaggerated. It is necessary to have some knowledge of the muscles involved, to limit treatments to those muscles which should be treated in this manner, and to see that the voluntary movements when they can be produced are directed to improvement in the movements which are in need of improvement. Elaborate apparatus is not needed, for much may be done with common objects which are to be found in any household. The principal requirement is a knowledge of the structures involved and their functional relations, with some ingenuity in the devising of means of bringing about movements of certain characters which should be practiced, and also different means which may help to stimulate the patient's interest and to show plainly to him the course of his improvement.

The facts are also of interest in a theoretical way in relation to our conception of motor cerebral control. If after the so-called motor cortex is destroyed, or the fibers in the internal capsule sectioned, by hemorrhage or by anemia, there is a possibility of return of motor control, we shall have to modify our conception of how the different parts of the brain act in the production of movement. Experimental and clinical results are at hand to indicate that there are great anatomic and functional

differences in different brains and that the relations of the motor cortex to the limbs are not so simple as they have been supposed to be. For recoveries of functions such as the authors have described there are many possible explanations. The simplest is that we deal not with motor inabilities but with exaggerated asthenias. This is an explanation of some of the motor deficiencies of animals from which the motor cortex has been excised. We should then look on the different forms of cerebral paralysis as exaggerated difficulties of movement and not as inabilities to move. The recoveries may also be explained as due to the assumption by another portion of the brain of the function of the motor cortex and the motor fibers. This was a favorite explanation of the motor recoveries of animals after ablation of the motor area on one side, it being assumed or concluded that the other hemisphere assumed the function of motor control on both sides of the body. That this explanation will not hold, in animals at least, is shown by the fact that animals which have recovered motor power of the limbs after the removal of the motor cortex of one hemisphere do not lose this motor ability if the motor cortex of the other hemisphere be subsequently destroyed. Another possibility is that the cerebral motor cortex may not be necessary for the production of movement but that this cortex is only regulatory. It has been shown that if the motor cortex on one side be excised, a considerable number of the pyramidal fibers at the medulla on that side do not degenerate, and it would seem that, assuming the pyramidal fibers to be wholly motor in function, some of the motor impulses may arise in portions of the brain other than the precentral cortex. The facts which they have already collected indicate that lesions of the motor cortex or of the upper part of the pyramidal tract in man do not abolish function, but put the function in abeyance until such time as the appropriate condition is present for the production of movement. We should probably not speak of permanent paralysis, or of residual paralysis, but of uncared-for paralysis; because many of the conditions appear to resemble, if they are not real, phenomena of disuse, rather than actual inabilities.

**Increased Sweat Secretion on the Paralyzed Side Following Pilocarpine Injection In Cortical Lesions.** G. Gikeles and J. Gerstmann<sup>1</sup> report a study of eleven cases of gunshot injuries of the skull. With one exception in which the lesion was located in the gyrus angularis, the injury was in the psychomotor region or nearly so. In seven cases of a group in which spasticity was absent, injection of 0.01 pilocarpine was followed in six cases by a stronger secretion of sweat on the paralyzed side. In five cases of a group with spastic phenomena 0.01 or 0.02 pilocarpine caused a more marked secretion on the paralyzed side.

Inasmuch as the upper extremities and the face showed this difference a greater number of times and to a more marked degree, the authors conclude that the psychomotor region, especially of the face and upper extremity lies in close relationship to the sweat center.

**Visual Disturbance from Wounds of the Head.** Among 300 cases of cranial wounds on the trench battlefields, Pierre Marie and Charles Chatelin<sup>2</sup> observed thirty-one with marked changes in the visual fields due to lesions of the visual cortex or intracerebral visual pathway. Most frequently they found homonymous hemianopsia from injury to the optic radiation in the deep portions of the temporal lobe, generally associated with severe destruction in the parietal lobe. In other cases, penetrating bullet wounds had involved the optic radiation or cortical visual area in the occipital lobe but hemianopsia from cortical lesion in one occipital lobe alone was uncommon, both sides being more frequently attacked. Complete hemianopsia usually means a lesion in the optic radiation, and on the other hand, there was no case of partial loss of fields from lesion of the optic radiation. Right hemianopsia was nearly always associated with more or less sensory aphasia. Hemianchromatopsia was only noted in one case, and the authors were surprised not to find it in any of the cases with only scotomata or less of one quadrant of the field. There were two cases of almost total blindness from lesions of

(1) Neurol. Centralbl., October, 1915.

(2) Revue Neurol., November-December, 1915.

the cortex of both occipital lobes with subsequent partial return of vision.

Horizontal inferior hemianopsia is a rare condition in times of peace and has usually been associated with a lesion of the optic chiasma. It was not infrequently seen by the authors in cases of wounds above the external occipital protuberance and was evidently caused by injury to the upper lips of both calcarine fissures. Horizontal superior hemianopsia was never observed, probably because injury to both lower lips of the calcarine fissure would almost certainly be associated with injury to the cerebellum and the dural sinuses and be immediately fatal. There were two cases of homonymous quadrant hemianopsias, one ascribed to lesions of the upper lip of one calcarine fissure, the other to lesions of the upper part of one optic radiation. Furthermore, there were five cases of hemianopic scotomata, macular and paramacular, three of purely macular scotomata and four of multiple scotomata. In most of these cases there were shell wounds in the vicinity of the occipital protuberance with penetration of pieces of shell or of the internal table into the brain. There was usually at first complete blindness for a few hours, with rapid partial return of vision. Sometimes there were also at the time of the injury symptoms of irritation of the visual cortex, such as flashes of light like exploding fireworks, and in one case the picture of ophthalmic migraine with scintillating scotoma has persisted for several months.

Numerous cases of wounds of the occipital lobes without any alteration in the visual fields were observed but in these cases only the external portions were affected.

**New Infantile Form of Diffuse Brain Sclerosis.** The so-called diffuse scleroses of the brain in children are divided by K. Krabbe<sup>3</sup> into at least three quite distinct types: (1) a syphilitic form; (2) Schilder's encephalitis periaxialis diffusa; (3) a familiar infantile form, of which five cases are discussed in this paper. The literature probably contains one other case. This form shows the following characteristics: it is usually a familial disease; it sets in somewhat acutely in about the fifth month in a child who up to then has been quite healthy; it pro-

(3) Brain, June, 1916.

gresses on a chronic course, ending with death from five to six months after the onset; universal rigidity of the musculature, violent tonic spasms, probably causing pain, and brought on by touching or noise, form characteristic symptoms. As a rule, nystagmus is present, and in the latter stage atrophy of the optic nerve. Periodic elevations of temperature occur without perceptible cause, outside the central nervous system. Finally, extensive paresis and pronounced debility close the scene.

The pathologic anatomic findings are: A marked hardness of the white substance of the brain without alteration of its shape. Microscopic examination of three cases showed relative intactness of cortex and the basal ganglia, the nervous centers of the brain and of the spinal cord; destruction of the medullary sheaths and axis cylinders throughout the white substance of the cerebrum (a 2 mm. layer, however, is preserved immediately under the cortex). Complete destruction of the white matter of the cerebellum and degeneration of the spinal nerve tracts are present. The destroyed tissue is replaced by dense fibrillar glia, in which are seen a considerable number of variously shaped glia cells, mostly protoplasmic; the vessel sheaths are infiltrated with fatty granule cells and other apparently gliogenous scavenger cells. There is a total want of new formation of vessels or infiltration of the vessel sheaths with plasma cells, lymphocytes or leukocytes. Krabbe is of the opinion that the affection must be regarded as purely degenerative and not as an inflammatory process. The disease presents a certain relationship to Pelizaeus-Merzbacher's disease, aplasia axialis extracorticalis congenita, on one side, and to Tay-Sachs' form of familial amaurotic idiocy on the other side. In other respects, however, it differs conspicuously from both these groups.

**Fatty Degeneration of Cerebral Cortex.** From the data provided by his investigations, H. A. Cotton<sup>4</sup> concludes that in all pathologic processes of the cortex which end in dementia and death, the fatty degeneration of the elements of the cortex plays a not unimportant part. The characteristic change for most of the psychoses is found in a great increase in amount of fatty deposits

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(4) Jour. Exp. Med., October, 1916.

when compared to normal individuals of the same age. In some processes such as senile dementia and dementia praecox the fatty substance appears to fill the cell body completely, and these cells have apparently lost all their functioning power. It is not common to find the fatty deposits in the processes of the ganglion cells except in dementia praecox, and to a limited extent in senile dementia. In other cases the pathologic variety of the fatty deposits in the ganglion cells is seen to be diffused over the whole ganglion cell. Cotton was able especially to observe this in infectious psychoses, in general paralysis, and in epilepsy. The so-called neuritis assumes a peculiar attitude in that it plainly leads swiftly to an acute fatty degeneration of the ganglion cells, in which there exists an inclination of the fatty granules to flow together into large masses. Frequently the fatty degeneration of the ganglion cells appears to be connected with the sclerosis of the cells, especially when it is a matter of slowly progressing alterations of degeneration. In the young, chronic cases of dementia praecox, far-reaching fatty degeneration of the ganglion cells, especially in the second and third cortical strata, likewise occurs. These findings should constitute an important contribution to the pathologic anatomy of dementia praecox.

#### TUMORS OF THE BRAIN.

**Sarcomas of the Base of the Skull.** Among the intracranial tumors, giving rise usually to symptoms of increased intracranial pressure and sometimes to extracranial signs and symptoms, are found a certain number of sarcomas.

C. E. Royce<sup>5</sup> says that the sarcomas found among the intracranial tumors may originate inside the brain; in the pituitary substance; in the walls of the vessels; in nerve sheaths, or in the bones and periosteum of the cranium. In this article he takes up only those arising from the bones of the base of the skull.

The reported cases of sarcoma of the bones of the base of the skull are not numerous. In 1909, Joseph Smith reported two cases of post-traumatic sarcoma of the skull

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(5) Jour. Amer. Med. Ass'n., April 22, 1916.

and reviewed the cases collected by Weisswange and Frohking. The combined series of the two latter writers amounts to 136 cases. Weisswange's paper was written in 1897.

Royce has found reports of twenty sarcomas of the bones of the base of the skull. Of these, twelve occupied the middle fossa; two the posterior fossa, and one the middle and posterior fossae.

Referring to Hartmann's paper Royce continues:

"In general he has to say of the tumors of the base of the skull that they occur widespread along the middle or posterior fossa, and may involve the middle and posterior or posterior alone." Royce also quotes Seeligman's remarks concerning tumors involving both middle and posterior fossae. "They are supposed to develop extradurally, and rarely progress toward the brain from the dura, but invade the bone and sometimes send processes into the adjoining cavities. Because the tumors gradually grow about the nerve roots, gradually disturbances of the cranial nerves will appear."

Concluding, Royce says that in general, these tumors are well encapsulated and separated from the brain substance by pia-arachnoid, altering the brain substance by pressure changes only. The cranial nerves sometimes give the only evidence, as in those growths involving the Gasserian ganglion. Frequently, however, there is extracranial evidence appearing in the nasopharynx, the hollow portions of the bones adjoining the nasal cavity, the ear or in the metastases, especially to the cervical lymph nodes.

**Report of a Case of Central and Peripheral Neurofibromatosis.** Peter Bassoe and Frank Nuzum<sup>6</sup> record the case of a boy 15 years old. The parents and their three other children were well. The maternal grandmother is said to have had a lump on the head and a maternal uncle has a small nodule on the forehead, but enough facts could not be determined to learn whether these really were cases of molluscum fibrosum. The first evidence of his nervous disease was in 1904, when at the age of 5 the boy had an attack of pain in the right scapular region suspected of being in the nature of pleurisy

(6) Jour. Nerv. and Ment. Dis., December, 1915.

but it was followed by a dragging of the left foot. From that time on he had similar attacks of pain in the right scapular region lasting from two weeks to three months and occurring about once a year. In 1909, eight eye-muscle operations were performed for the relief of strabismus. A lump on the left side of the neck was noted at least three years before death, others more recently, while a large mass attached to the rectum had been discovered only a few weeks before examination.

The final attack of illness began on Dec. 20, 1913, like the previous ones with pain beneath the right scapula, which lasted for about two weeks and was followed by pain in the left side of the abdomen, difficulty in emptying the bladder, and pain and progressive weakness in the left leg. On account of the scoliosis, x-ray examination of the spine was made and was negative. About March 1, occasional choking during eating set in and the pulse-rate became accelerated, being usually above 100. The boy was bright and showed no mental abnormality at any time.

*Examination on March 23, 1914.*—"The boy is very emaciated and exhausted and can not talk above a whisper. Subcutaneous, movable nodules are seen on the left side of the forehead, on the left side of the neck and on the chest. There is a well-marked dorso-lumbar scoliosis with convexity to the right. The patient can not raise his head from the pillow and the left leg is very weak. Passive movements of the lower extremities are painful. Smell and taste are normal and there is no important visual defect. The right external rectus is weak and the patient sees double when looking to the left. The corneal reflexes are absent and there is distinct paresis of both divisions of the left facial nerve. Hearing is much impaired in the left ear but good in the right ear. Ophthalmoscopic examination is negative. The left knee-jerk is absent and the right one weak. The ankle-jerks and the cremasteric and abdominal reflexes are absent. The Babinski sign is present on the right side, while the left plantar reflex is normal. Tactile sensation is normal but pain and temperature senses are diminished on the anterior aspect of the left leg and in areas on the abdomen."

The patient gradually grew weaker and the choking

attacks became more frequent. By the end of April there was evidence of increased facial nerve weakness on both sides, as both eyes would remain open.

The patient died on May 25, 1914.

The necropsy was held twelve hours after death.

*Anatomic Diagnosis:* "Multiple central and peripheral neurofibromata. Marked emaciation. Hypostatic hyperemia of the lungs. Passive hyperemia of the linings of the alimentary tract, urinary bladder, of the liver and spleen. Slight fatty changes in the kidneys. Accessory lobe in the left lung. Deep sacral decubitus.

"The brain weighs approximately 1,400 gm. There is a moderate edema of the pia over the vertex and over the temporal lobes. The pia is everywhere smooth and glistening and is not thickened. There is a firm, fibrous tumor in the left middle fossa, 4 by 3.5 by 2 cm. in its dimensions, which is intimately connected with the Gasserian ganglion.

"There is a similar tumor in each cerebello-pontine angle, each the size of an English walnut. These tumors have caused depressions in the cerebral and cerebellar hemispheres which come in relation with them.

"There is a small nodule 0.4 cm. in diameter connected with the left third nerve. There are numerous nodules varying in size from a split pea to a lima bean, intimately connected with the roots of the spinal nerves. There is one large tumor 8 cm. long and 2 cm. in diameter in the cauda which has caused marked pressure on the terminal filaments of the cord.

There are three nodules connected with the left vagus nerve, the smallest pea size at the level of the hyoid bone and the largest the size of a hazel nut, this being the nodule described just above the left clavicle. The third nodule is lima-bean-sized and is adherent to the larger nodule just described" (see Plate III.).

*Histologic Examination:* "The tumors present the same essential structure everywhere; namely, that of fibroma, often with a very whorl-like arrangement of the fibrous tissue. No new-formed nerve fibers are seen but the Pal-Weigert method reveals the persistence of old ones in some of the tumors, especially those of the cerebello-pontine angles."

Cases like the one just reported are now fairly numerous in the literature and on the whole rather similar. The number of tumors within the cranial and spinal cavities is usually large. The experience of Hunt and Woolsey of finding only one tumor in the spinal canal in a case of cutaneous neurofibromatosis is unusual. The fifth and eighth cranial nerves are those most frequently involved within the skull but when we include all the peripheral nerves the order of frequency seems to be: vagus, abdominal sympathetic, sciatic. The intraspinal tumors usually rise from the roots, especially the posterior ones, and when the cord is invaded it is usually in the region of the entry of the posterior roots. The thoracic region of the cord seems to be the favorite site, but the largest tumors are apt to be on the roots of the cauda.

**A Mixed Tumor (Chondro-Fibro-Epithelioma) of the Chorioid Plexus.** W. H. Burmeister<sup>7</sup> reports the presence of a tumor of the chorioid plexus in a male negro about 60 years old. Microscopically, the papillae were found to possess a multilayered epithelium. The basal layer consisted of low columnar epithelium having a distinct palisade arrangement and being sharply defined from the superimposed layers, the cells of which were markedly flattened. The cell outlines were indistinct, the upper cell layers appearing more as a syncytial mass. The nuclei of the lower layers stained deeply and were plump, vesicular and centrally located. The nuclei of the upper cell layers were very much flattened and distorted. The basal portions of the crypts between papillae were frequently completely filled with the epithelium. The older layers of the central portions of these alveoli were not infrequently necrotic, forming a pseudo-glandular structure filled with glandular cell-debris. They resembled very much the so-called cylindromata of the brain except that the pseudo-lumina contained a granular and not a hyaline-appearing substance. In the neighborhood of the necrotic areas, which frequently were strikingly regular in outline, the cell nuclei were markedly pycnotic. The cytoplasm of the cells stained with varying grades of eosin to almost a bright carmine, the most

(7) Bull. Johns Hopkins Hosp., December, 1915.

brilliant eosin appearing in the most markedly degenerated areas. The epithelial cytoplasm was very finely granular, and non-vacuolated. There was no evidence of any tendency to hyaline epithelial degeneration. In the degenerated portions the nuclei had undergone a finely granular fragmentation by karyorrhexis. Occasionally the cells of an entire alveolus were seen to be necrotic, or a group of alveoli had undergone necrosis. There was no evidence of an infiltrative growth. Mitotic figures were very few; not more than one in sixty to 100 fields were found. Weigert's neuroglia and Mallory's phosphotungstic-acid hematoxyline stains revealed no tendency to the formation of neuroglia fibrils, as is sometimes seen in the epithelium of true ependymomata. The stroma of the papillae consisted of a finely fibrillar, apparently edematous, connective tissue, whose cells were polygonal, branching and occasionally vesicular.

In those planes in which the capillary blood-vessels had been met with, the blood space was centrally located and separated from the adjacent stroma by only its endothelial lining. Irregular necrotic areas were frequently found in the stroma of the body of the tumor. These consisted of an eosin-staining granular debris and usually contained small calcareous granules with a typical concentric lamellation ("brain sand").

The body of the tumor consisted for the greater part of islands of typical hyaline cartilage, each island having a definite perichondrium of more compact connective tissue than that of the stroma generally. The cartilage throughout was intact, with no tendency toward necrosis or calcification (see Plate IV.).

Burmeister has not been able to find a similar chorioid-plexus tumor described. The growth, it would seem, could best be classified as a mixed tumor—a teratoma, the mesoblastic cartilaginous elements of which were carried in at the time of the embryonic pushing-in of the velum interpositum of the brain, leading in part to the formation of the chorioid plexus. That we were dealing with a pure fibro-epithelial tumor with a metaplastic transformation of its connective tissue to cartilage would seem less likely.

**Convulsive Spasm of the Face Produced by Cerebello-pontine Tumors.** During the past two years examples of facial spasm of unusual persistence and severity have been seen by Harvey Cushing<sup>8</sup> among the brain tumor cases in his clinic. One of them had been previously diagnosed as focal epilepsy ascribed to a lesion of the contralateral cortex. Both proved to be peripheral irritation of the facial nerve from pressure of a homolateral tumor of the cerebellopontine.

Cushing reports the case of a man 30 years old who for nine years had been troubled by *twitching of the left side of the face*, beginning as a blepharospasm of mild degree coming on a few times a day and lasting for a few seconds. There had been gradual increase in the number, intensity, and surface extent of these spasmodic movements, until during the previous six months they had been very severe, coming on every ten minutes or so and persisting for a full minute or two. The spasms began with a twitching and drawing of the muscles about the eye and gradually the whole left side of the face was pulled over in a grimace and the surface muscles of the neck were affected. The man stated definitely that there had been a few severe attacks in which the left arm and leg had mildly participated in the attack and that the left arm and hand had felt numb on several occasions.

For three years he had had occasional momentary shooting pains in the frontal region produced by sudden movements and for two months there had been some sub-occipital discomfort on arising in the morning, but he did not regard these as *headaches*. Vision had been poor for many years and he had had repeated changes of glasses. For a year there had been definite blurring of objects, a good deal of photophobia, and occasional diplopia, but he retained reading vision. Hearing on the left had been poor for many months and was ascribed to former attacks of "earache."

He suffered for a year from some "indigestion" and from constipation, and for two weeks had had occasional morning vomiting which was blood-tinged at times. For some weeks, too, he had had some difficulty in swallowing, which required an extra effort; talking was also some-

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(8) Jour. Nerv. and Ment. Dis., October, 1916.

what difficult and he had had repeated attacks of hiccoughs. He had also had a good deal of nasal catarrh and several nose-bleeds.

For a year he noticed an unsteadiness of gait with occasional staggering. This increased to such an extent that for six months he had been unable to walk. He attributed this entirely to dizziness.

On several occasions during the first six months he had olfactory hallucinations as of something burning (rags). There was some confusion at the time and the sensation might last for five or ten minutes. No gustatory sensation occurred.

He saw many doctors, most of whom regarded his trouble as of gastro-intestinal origin. He lost over twenty-five pounds in weight.

The positive findings as assembled were as follows:

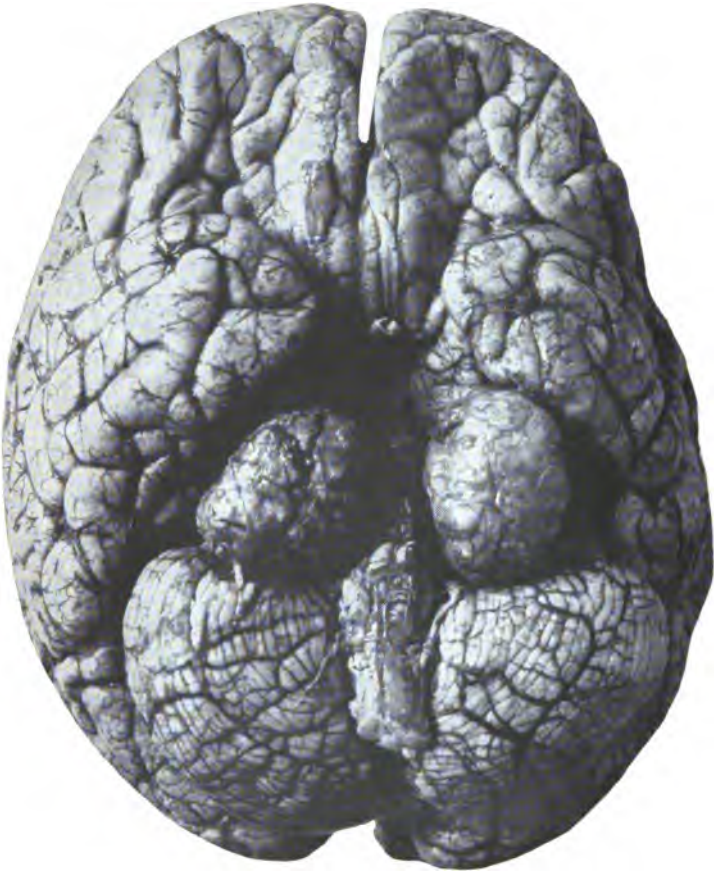
*Subjective.*—"Twitching of the left face (9-10 years). Intracranial pains (18 months). Gastric disturbances and failing vision (1 year). Some dysphagia and dysarthria (5-6 months). Dizziness."

*Objective.*—"Left facial spasms. Bilateral choked disc, more advanced on right. Right parietal tenderness. Weakness and slight hypesthesia of left side of body. Diminished superficial and exaggerated deep reflexes on the left with ankle clonus and positive Babinski. Slight ataxia of extremities. Slight dysarthria and dysphagia. Nystagmus."

Subsequent and repeated studies brought out the following additional points and certified the diagnosis of a subtentorial lesion.

*Special Cerebellar Studies.*—"Head held somewhat tilted to right shoulder. Flexing and twisting of head causes increase in general discomfort. Definite nystagmus, both lateral and vertical. Corneal reflex relatively inactive left. Slight hypesthesia left face. Jaw deviates slightly to left and left masseter weaker than right. Taste discrimination poor on left. Emotional movements of left lower face less good than right. Loss of hearing on left (apparent bone conduction probably transferred as it is lost with caloric test). Definite ataxia of left arm and leg with dysmetria (gives a history of clumsiness of left hand and general enfeeblement of left side before

PLATE III.



Base of brain showing tumors in both cerebello-pontine angles.



Large tumor of cauda equina.

Case of central and peripheral neurofibromatosis.—Bassoe and Nuzum (see par



PLATE IV.



Mixed tumor of choroid plexus. Section through both cerebral cortices. The section of the left cortex was made at a level 2 cm. below that of the right cortex. The section through the right cortex also passes through the tumor in a plane at the juncture of its upper and middle thirds, leaving its pedicle-like attachment to the choroid plexus intact.—Burmeister (see page 93).



confinement to bed). Caloric tests: no response left, normal right. Diadococinesia: poor right; less good, left."

Following operation, the patient made a good operative recovery, the wound healed without reaction (Figs. . . and . .). The facial spasms gradually subsided together with all other symptoms except his nystagmus, which persisted. There was even a considerable restoration of hearing on the left. The choked disc cleared without further lowering of vision.

Cushing summarizes the case as follows:

"This paper deals with a case of typical convulsive spasm of the left face which after many years' duration became associated with the evidences of intracranial pressure. The combination of symptoms might easily have been mistaken for cortical epilepsy due to a cerebral growth, whereas they were apparently produced by the pressure against the facial nerve of a subtentorial tumor in the lateral recess.

"The recurring spasms were absolutely painless and subsided after the operation and partial removal of the tumor, a procedure which must have served to relieve the pressure against the nerves, for even the severely implicated acusticus regained some of its function. It must be admitted, in view of the histologic nature of the tumor (glioma), that the facial spasms may have antedated the growth though, all things considered, this seems improbable."

**Symptomatology and Diagnosis of Intracranial Tumors of the Middle and Posterior Cranial Cavities, Growing from the Region of the Gasserian Ganglion and the Cerebello-Pontine Angle.** Williams B. Cadwalader<sup>9</sup> records the observations made on nine cases of tumor occupying the so-called cerebello-pontine angle or region of the Gasserian ganglion. He says that the most important differentiating signs in tumors of the Gasserian ganglion appear to be severe sensory disturbances, both subjective and objective, of the face on the same side as the lesion; sympathetic paralysis of the eye on the same side, third nerve paralysis, disturbances of smell, unilateral visual disturbances on the side of the growth,

(9) Jour. Nerv. and Ment. Dis., July, 1916.

and ataxia. All these signs may precede the development of deafness; or deafness may be absent, and none of these signs except the ataxia are common symptoms of tumor of the cerebello-pontine angle.

The importance of recognizing these different types of new growths become obvious if surgical interference is contemplated.

In conclusion, he calls attention to the fact that there is a particular type of tumor that grows from the dura at the base of the brain in the middle and posterior cranial fossae which frequently involves the bones of the skull and does not infiltrate the brain substance, but does embrace the cranial nerves. These tumors are endotheliomata and are sometimes associated with cystic formations. They may arise from the region of the Gasserian ganglion and extend backward as a flat, slow-growing mass and subsequently involve the seventh and eighth nerves, thus producing symptoms that resemble the true fibromatous tumors which always arise from the eighth nerve in the cerebello-pontine angle.

#### ABSCESS OF THE BRAIN.

**Report of Case in an Infant.** Holmes<sup>1</sup> reports a case in an infant, 23 months old, that proved to be multiple abscesses of the brain, and that, clinically, was practically undifferentiable from chronic internal hydrocephalus. Seventeen days following an exploratory craniotomy the child died.

Post-mortem examination revealed a large abscess in the white matter of the left hemisphere. A smaller abscess lay mesial to the posterior extremity of the first abscess and projected backward and outward into the occipital lobe. Three smaller abscesses were discovered in the tips of the occipital lobe. The aqueduct of Sylvius was obstructed and an internal hydrocephalus was present. Microscopic examination showed unusual development and sharp definition of the fibrous capsule, or zone of connective tissue reaction and prominence and great variety of the "cerebral granule cells" or "Gluge's" corpuscles. The etiology of the abscess was

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(1) *Archiv. Int., Med.*, May, 1916.

**PLATE V.**



**Case of multiple abscesses of brain in infant. Sagittal section of brain showing abscess. Two-thirds natural size.—Holmes (see page 98).**



undetermined. It was not possible to exclude multiple tubercles which had undergone puriform softening (Plate V.).

**Late Abscesses After Gunshot Wounds of the Brain.** Otto Marburg and Egon Franze<sup>2</sup> say that from among the great number of men with gunshot wounds at the clinic, during the first half year of the war, sixty-two were operated on.

It happened that among some of these patients, who were in a seemingly fair condition, there appeared a disease which despite all means brought on death in a few days.

Anatomically there were found in such cases encapsulated abscesses which remain dormant a long time and which manifest themselves without any cause. The time in which these abscesses made themselves evident ordinarily was four or five months after the first operation. In one case the interval was more than eight months.

The symptomatology of the late abscesses is quite characteristic. With the rise of the temperature there arise general symptoms (headache, vomiting), to which in twenty-four hours are added "irritative phenomena" and these are always the symptoms of the beginning of meningitis, stiffness, Kernig's sign, general prostration with changeable consciousness and the pulse responding to the fever. That everything is not quite normal is shown by the numerous localizing symptoms appearing as hemiplegia, aphasia, etc. It seems important that Jacksonian attacks happened only in two cases. In one at the beginning and in the second case only once, so that they can by no means be marks of a progressive brain process. So when a shotgun wound with an injury to the brain is operated on with success and a certain time afterward there is rise in temperature, meningeal symptoms set in and the focal symptoms show a small aggravation, the authors assume the existence of late abscess. Where do the symptoms come from? Manifestly from the propagation of the pus in the meninges. The reason is plain; often the small abscess is joined through a hardly noticeable gap with the ventricles, there appears a pyocephalus and the pus then runs through the foramen

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(2) Otto Marburg and Egon Franze, Neurol. Centralbl.

Magendi to the meninges at the base of the brain and from there to the hemispheres.

### THE BASAL GANGLIA.

**Report of Two Cases of Progressive Lenticular Degeneration.** Arthur S. Hamilton and Herbert W. Jones<sup>2</sup> report two cases which though not in all respects typical of chronic lenticular disease as outlined by Wilson they believe to belong to this group. The condition is clearly familial but not, so far as determined, hereditary.

Cases 1 and 2 were evidently of the chronic type and, at the time of the report, had lasted eight and about two years, respectively. The average duration of eight chronic cases, as given by Wilson, was almost exactly four years. In three acute cases, the patients died at four, six, and seventeen months respectively. Dr. Homen's case lasted seven years. Cassirer's had lasted thirteen years. Sawyer's case (accepted with some reservation by Wilson) had lasted seventeen years. Cadwalader's second case had lasted twenty years and Strümpell's case had lasted twenty-eight years at the time the reports were made (see Plate VI.).

In respect to the motor phenomena the authors' cases are not in entire agreement with Wilson's description, although here again there has been considerable variation in the symptoms described in certain recent cases. Tremor was present in both of their patients but was hardly so persistent or widespread as it appears to have been in most of Wilson's cases. Moreover, in Case 1 this tremor grew less in the later stages of the disease, in which respect it is in agreement with Sawyer's, Cassirer's and Strümpell's cases, though opposed to the principle laid down by Wilson that "as the disease progresses, the tremor, according to the experiences of all the observers, becomes worse in every way."

As has been true in all cases observed by others, hypertonus has been a very pronounced feature. In the second case it has increased steadily as the authors observed the progress of the disease and at all times has been prominent in both cases, but it has seemed to

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(2) Nerv. and Ment. Dis., April, 1916.

PLATE VI.



Case of **progressive lenticular degeneration**.—Hamilton and Jones  
(see page 100).



diminish somewhat in Case 1, in the later stages; and, at the present time, there are periods when, in the arms, it disappears and the arms become, for a few moments, even hypotonic. This is contrary to Wilson's experience but was true of Sawyer's case and in Cassirer's and other cases there were distinct changes in the degree of stiffness at different times.

Though contractions are pronounced in Case 1 and very distinct in Case 2, no definite contractures have developed up to the present.

Dysarthria and dysphagia have reached an advanced stage in Case 1, and are fairly well developed in Case 2, and evidently still increasing in the latter, but in neither is there complete paralysis of the palate.

Probably in no respect do these cases diverge so greatly in important features from Wilson's description as in the evidence of pyramidal disease. Wilson has shown both clinically and pathologically that the motor involvement is essentially extra-pyramidal. In both these cases the motor symptoms were clearly extra-pyramidal when first seen, but in Case 1 the abdominal reflexes were found to be very greatly diminished in the spring of 1913, and, on one occasion, during the following summer, both plantar responses were clearly extensor, though repeated attempts previously and afterwards, always gave a flexor response. At the time of writing this article (1915) an extensor response is frequently obtained in the left foot but never in the right. In the second case, approximately one year after the examination, the left abdominal reflexes were faint and the plantar reflexes were uncertain, sometimes flexor and sometimes extensor.

Nystagmus has been present in both the authors' cases at times, contrary however, to all other reported cases.

Emaciation and muscular weakness are symptoms referred to by Wilson as common and significant. Neither has been present in any pronounced degree in their cases but this may be because both belong to the group of chronic cases and neither has yet advanced sufficiently far.

A possible pathologic and even clinical relationship

between chronic lenticular degeneration and paralysis agitans has been often described, and Strümpell, in a recent article, announces his belief that paralysis agitans, pseudo-sclerosis and Wilson's disease all belong to the same group. Paralysis agitans, without agitation, he regards as particularly like Wilson's disease.

That these two cases may not belong with the Westphal-Strümpell type of pseudo-sclerosis is by no means so clear, and the more the cases are multiplied under these two headings the more difficult does the distinction become. Several cases are now on record, generally accepted as pseudo-sclerosis, where the autopsy has revealed a very definite lesion of the lenticular nucleus though none in which the changes in this region have been so pronounced as in Wilson's cases or in which the changes were so clearly limited to the lenticular and subthalamic regions. The liver appears to be in much the same condition in the two diseases but in the cases of pseudo-sclerosis a large amount of pigment has been described in the internal organs and this has also been found clinically in the outer ring of the cornea. Assuming that there may be a clear differentiation between Wilson's disease and pseudo-sclerosis, it would appear that the early and marked mental disturbance insisted on so strongly by Strümpell, in the latter, together with the hemi-paresis and paraparesis and the corneal pigmentation, all argue strongly against the admission of these cases.

**Pseudo-Sclerosis, Wilson's Disease and Associated Diseases.** (The Amyostatic Symptom-Complex.) Adolph Strümpell<sup>4</sup> says that the amyostatic symptom-complex occurs in cases of different diseases, which are apparently very closely related to each other in anatomic-physiologic causes, although they may present great differences from the etiologic standpoint. From this standpoint the infantile or juvenile, frequently hereditary or familial types are different from the forms which appear less frequently in older persons. We have on the one side the so-called pseudo-sclerosis, Wilson's disease, on the other a disease picture which is related to paralysis agitans; many of the cases known

(4) Deutsch. Zeitschr. f. Nervenhlk., December, 1915.

under the name of juvenile or hereditary or paralysis agitans probably belong to the first group. In the two groups the disease pictures are classified according to the predominance and grouping of the single symptoms. The symptoms to be considered are first, the tremor athetoid movements; second the muscle rigidity (static muscle rigidity) with its accompanying symptoms (contractures, clumsiness, etc.); third, speech disturbance often in connection with dysphagia; fourth, psychical disturbances, slowly progressing dementia; fifth, the accompanying liver disease and pigmentation of the cornea.

Familial infantile types occur with tremor muscle rigidity, speech disturbance, liver disease and corneal pigmentation; and on the other hand are types in which muscle stiffness predominates almost without tremor, or dementia and without a pigment ring on the cornea. In case of diseases which appear in older people we have a typical paralysis agitans with tremor and muscle rigidity but without special speech disturbance or dementia. Besides this we have cases of so-called paralysis agitans *sine agitatione*, such as "arterio sclerotic, muscle rigidity, etc., with predominating muscle rigidity and often with dementia but without tremor." These cases Strümpell likes to call "myostasy." Nothing is known of the state of the liver or of pigment anomalies in these cases.

Isolated amyostatic symptoms are found in other nervous diseases. In the future when disturbances of the motility are observed we should give consideration to the differentiation of pyramidal tract diseases and the amyostatic symptom-complex.

**Pathology of the Corpus Striatum and Extra Pyramidal Motor Disturbances.** W. M. Van der Scheer and F. J. Stuurman<sup>5</sup> report a case of a man with a tumor of the corpus striatum, involving the head of the caudate nucleus and infiltrating the lenticular nucleus. Reviewing the opinion of Klust they say that the ataxic hypotonic choreiform symptoms of the ascending cerebello-cortical tracts are contracted to the descending cortico-pontine-cerebellar tracts, interruption of which

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(5) Zeitschr. f. d., ges. Neurol. u. Psychiat., November, 1915.

frees certain reflexes, for which the cerebellum is an intermediary agent (the attitude and position reflexes, the reflexes of the proprioceptive system of Sherrington) from inhibiting influences. A similar condition must occur if the origin of this tract is injured at the cerebral cortex. In such a manner, forced attitudes could be explained in frontal cortical lesions.<sup>6</sup>

Paralysis and severe psychoses with disturbances of motility and perhaps the tonic symptoms (tension flexibility) in the psycho-motor paralysis (akinesia) may be similarly explained; from this view Wilson takes a dissenting attitude.

The authors' case in which a lesion appeared in the transcortical connecting paths seems to prove that "tonic innervation" could occur in cases of subcortical disturbances in which a bilateral lenticular softening is formed. Of considerable interest in this connection is their mention of incontinence of urine which they explain on the basis of absence of antagonistic atonic impulses and inversely the contraction of the agonists applied to the bladder musculature as to the general muscular system.

**Progressive Atrophy of the Globus Pallidus. A Special Form of the "Paralysis Agitans Syndrome" Occurring in Early Life and Associated with Atrophy of the Cells of Globus Pallidus.** Dr. J. Ramsay<sup>7</sup> states that paralysis agitans undoubtedly includes a variety of types which are related clinically but which must present differences in the localization and character of the underlying pathologic lesions. In the present study an effort was made to isolate a special group of paralysis agitans cases, namely, the juvenile type, on the basis of certain definite changes in the motor cells of the corpus striatum. In one case which was studied, the sole lesion was atrophy and disappearance of the large motor cells of the globus pallidus system. The globus pallidus system is the motor center proper, while the neostriatum (caudate and putamen) forms an inhibitory and coördinating cortical mechanism.

(6) The articles of C. K. Mills and J. H. Lloyd on this subject are reviewed in the Practical Medicine Series, 1915, Vol. X, pp. 92-101.

(7) Proc. Amer. Neurol. Soc., 1916.

The mechanism which is involved in this disease is the motor or afferent system of the globus pallidus. A destructive lesion in the case of the globus pallidus produces not only paralysis of certain automatic and associated movements, a slowness of movement and loss of motor initiative but a great increase in muscular tonus as well. An affection of the small-cell system of the neostriatum releases the motor mechanism of the globus pallidus from control, and there result the phenomena of the chorea of Huntington. A destructive lesion of both types of cells produces chorea, athetosis, spasms, rigidity, and tremor in various combinations.

**Pathologic Findings in Paralysis Agitans.** The pathologic changes noted in the region of the basal ganglia in the two cases studied by E. W. Auer and S. P. McCough<sup>8</sup> were:

1. Areas of rarefaction containing neuroglia cells and debris giving the tissue a moth-eaten appearance.
2. Clean punched-out holes possibly excessively enlarged perivascular spaces from which the vessels may have dropped out.
3. Round and oval basic-staining deposits chiefly in the perivascular space and adjacent tissues.
4. Diminution in the number of the external medullary lamina and of the radial fibers of the lenticular nucleus with some evidence of degeneration of the latter.
5. Failure of the cells of the corpus striatum to stain well which latter may possibly have been due to the age of the material.

6. In one case advanced degeneration of the cells of the centrum medium on both sides and of the corpus subthalamicum.

**Report of a Case Illustrating the Anatomic Seat of Mobile Spasms.** Charles L. Dana and Belden Gere<sup>9</sup> report the case of a child, aged 6 years, born prematurely, normal until the fourth month; then an attack of stupor, etc., diagnosed as "meningitis," then development of clonic and tonic movements involving face,

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(8) Jour. Nerv. and Ment. Dis., June, 1916.

(9) Amer. Jour. Med. Sci., 1916.

throat, neck, trunk, arms and legs, the legs being mainly rigid, so that the child could only imperfectly stand or walk; no paralyses, no atrophies, no anesthetics, special sense normal, intelligence retarded but not seriously.

The duration of the condition was five and one-half years. Death followed operation to resect the posterior spinal roots.

Autopsy revealed no gross lesion of brain or cord; the brain was of normal size and weight.

Microscopically, the motor and premotor cortex showed disturbance in lamination of cells, degenerative changes of cells, deficiency in size and number of large pyramidal cells. Changes were less marked in posterior central convolution. Changes similar to those of the anterior central convolution though much less marked were found in the thalamic and subthalamic region. Nuclei of columns of Goll and one pyramidal tract showed some deficiencies.

The tonic and clonic spasms with some permanent rigidity of the lower extremities were the main features of this case. The tonic spasms caused an apparent spastic paraplegia, although there was no real paralysis, nor objective evidence of involvement of the pyramidal tracts, and both the corticospinal and the peripheral motor neurons showed no or slight signs of degeneration.

The case is important in that it shows an anatomic seat of such spasms as those of chronic chorea, spasmodic ties, and myotonia.

The authors state that the subject of cerebral hemiplegia and diplegia without involvement of the pyramidal tracts is discussed in full by Dr. H. Vogt. In these cases sometimes at least the spastic paralysis seems due to atrophy of the superficial layers of the cortex with intact cells of Betz. In their case it was not so much atrophy of particular layers, but various irregular displacements, degenerations, and focal atrophies. This would explain the irregular motor discharges.

"The cortical changes that were found are sufficient to explain the spasmodic symptoms, and as these

lesions were induced by some infection four months after birth it is a fair inference that they were primary and not secondary factors in the disease. We believe that they give us an indication of the pathologic anatomy of the chronic degenerative myoclonic and myotonic spasms.

"It seems to be fairly well established that the chronic, coarse, jerky, ataxic tremors are due to involvement of the cerebellar rubrospinal and extrapyramidal tracts. Such involvement is seen in paralysis agitans, multiple sclerosis, and midbrain and cerebellar disease.

"But the clonic and tonic spasms such as are seen in spasmodic tics, torticollis, hereditary chorea, and myoclonias especially are cortical and due to defects and displacements of the cells of the central and precentral convolutions.

"This view is supported by the fact that cortical changes are found in chronic and hereditary chorea, in myoclonus epilepsy and in certain localized degenerative tics.

"The case presented clinically some of the features of Little's disease, with clonic and tonic spasms, but there was in our case no real paralysis and no involvement of the pyramidal or extrapyramidal tracts, at all adequate to explain the essential features of the symptoms."

### THE HYPOPHYSIS.

**Three Cases of Hypophysis Tumor Resembling Tabes and Paresis.** Gunnar Kahlmeter<sup>1</sup> reports three cases in all of which the patients showed at some time during their illness such symptoms of tabes or paresis, that a diagnosis of these diseases would have been excusable. In the first two cases the x-ray revealed the true nature of the disease. It is probable that not only hypophyseal tumors, but other brain tumors as well, can produce such a picture, as Thierauch<sup>2</sup> reported: the case of a man of 49, with a gliosarcoma,

(1) *Deutschr. Zeitschr. f. Nervenhk.*, September, 1915.

(2) *Zeitschr. f. d. gesam. Neurol. u. Psychiat.*, 3 and 28.

who had attacks of convulsions with temporary paresis one of whose pupils reacted sluggishly and who because of a luetic infection twenty years before gave a positive Wassermann reaction on the serum.

Absence of the patellar reflex is rather ordinary in various brain tumors, sensory disturbances may be similarly caused. Pupillary fixation and ocular muscular paralysis as well as psychical disturbances may more likely be caused by hypophyseal tumors, and they of course produce optic atrophy in the absence of choked disc more frequently than other brain tumors. Impotence is likewise observed more frequently in hypophyseal growths.

#### THE PINEAL GLAND.

**Studies on the Pineal Gland.** G. Horrax<sup>3</sup> from experimental observation on the pineal gland concludes that total experimental pinealectomy is possible in guinea-pigs and rats. Pinealectomized male guinea-pigs show a hastened development of the sexual organs, manifested before maturity by a relative increase in size and weight, both of the testes and seminal vesicles, over control pigs of the same litter. Histologically, the testes and seminal vesicles of these animals, if taken before the age of sexual maturity, show a more advanced physiologic state than do the controls.

He further notes his clinical observations in three cases, one of which was certified in full, the other two presented clinical pictures suggestive of pineal gland dysfunction. This one exhibited adiposity and macrogenitosomea, the others showed unusual precocity of adolescence and overgrowth. The certified one presented precocious adolescence. There were intracranial symptoms and a diagnosis was made of tuberculous meningitis and xanthochromia of the cerebrospinal fluid. Autopsy revealed a struma of the pineal gland. He adds the following summary:

1. Extirpation of the pineal in young chickens and lower animals tends to hasten normal maturity.
2. Tumors of the pineal gland in children occurring

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(3) *Archiv. Int. Med.*, May 15, 1916.

before the age of puberty usually give rise to a syndrome characterized by precocious adolescence.

3. Feeding the gland substance to young animals is said to have the same effect as extirpation, but the observations are somewhat inconclusive.

4. A report of three cases of supposed pineal tumor, one of which was certified by necropsy, is offered as a further contribution to the study of this gland.

#### THE CEREBELLUM.

**Cerebello-Olivary Tract.** Karl Schaffer<sup>4</sup> reports a case which he thinks very plainly points to the existence of Koelliker's cerebello-olivary tract. This tract originates in the cerebellum, runs through the restiform bodies, then taking the tract of the pretrigeminal and peri-olivary fibers, goes with the latter through the olive into the interolivary level, proceeds to the raphe, the crossed bulbar olive and the lateral olives where it ends. If this tract is involved there occurs on the side of the cerebellar lesion a diminution in size of the restiform bodies and an atrophy of the pretrigeminal and peri-olivary, arcuate fibers on the side opposite to the lesion, marked sparsity in the medullary fibers of the olivary mantel and diminution of the medullary contents of the hilum of the olive. The cerebello-olivary tract runs with the crossed olivocerebellar tract, both appear as one bundle with paths of opposite direction. The olivocerebellar tract originates in the ganglion cells of the main olive, the cerebello-olivary tract ends in the terminal brushes about the same ganglion cells, so that it is apparent that in event of a common lesion of the cerebello-olivary the ganglion cells experience a fibrolysis of double origin. First a retrograde one as the result of lesion of the olivary-cerebellar tract, and second a transneuronal one on the basis of an injury of the cerebello-olivary tract. This explains the rapid and complete disappearance of the olivary ganglion in cerebellar lesions.

**The Ear Tests of Barany in Locating Cerebellar and Other Encephalic Lesions.** B. Alex Randall and Isaac H. Jones<sup>5</sup> call attention to the Barany tests in relation

(4) *Zeitschr. f. d. ges. Neurol. u. Psychiat.*, October, 1915.

(5) *Amer. Jour. Med. Sci.*, April, 1916.

to the broader aspects of diagnosis. They have examined 125 pathologic cases, the study being especially directed to the intracranial side of the subject. For this purpose the pointing reactions have far surpassed the nystagmic.

It is hardly necessary at this time to describe in detail the technique of examination. In brief, however, as regards the pointing reactions, the normal person is always aware of the location of his hand or finger in space when his eyes are closed; and he is able with it to find an object previously located by him, as, for example, the finger of the examiner held in front of him. With the ear stimulated, either by cold or hot water or air or by turning in a smoothly revolving chair, he is no longer able to find the finger, but points *past* it in a definite direction, either to one side or the other, above or below, depending upon the exact stimulation employed. These "past-pointing" reactions are absolute in the normal; their modification marks the abnormal and challenges us to find the explanation and locate the fault.

If the right ear is douched with cold water of a temperature of 68° F., the normal person points to the right; if the right ear is douched with hot water of 112° F. he points to the left. This is true, in both instances, also of the wrist, elbow, foot, head, and trunk. Or if the patient is turned in a rotating chair toward the right and then suddenly stopped, all the pointings will be toward the right, and *vice versa* after turning to the left. In each of these tests the head must be rigidly supported in the chosen position.

There has been so much needless confusion, however, in the descriptions of these tests, especially in stating the direction of the nystagmus in comparison with the past-pointing, that the authors suggest an absolutely reliable and easy method of remembering these facts. Thus, the usual routine methods are by cold water and turning: Now if to the right (that is either turning to the right or douching the right ear) everything is to the right—all the pointings, shoulder from above, shoulder from below, elbow, wrist, neck or trunk, and the falling reactions, all are to the right. Now the deviation of the eyes, just as all the other reactions, is also to the right; but

this is the slow movement of the eyes to the right. The "recovery," the quick movement to the left, however, the cerebral component, is more conspicuous, and therefore this phenomenon has been called "nystagmus to the left."

They have come to a definite conclusion that all pointing reactions are primarily *cerebral* and not cerebellar, as generally stated. When one of themselves was tested and, for example, with erect head was turned to the right, he had on stopping a subjective sensation of turning to the left. For this reason he felt that he was leaving the finger of the examiner behind him and pointed to the right, where he conceived the finger to be. He deliberately pointed to the right unless by a non-automatic, calculated correction he overcame the misconception of the position of the finger. If after the usual past-pointing the examiner's finger were moved out to meet that of the examined, who then repeated the test, he past-pointed almost as much farther to the right, and would continue perhaps to the completion of an entire circle in his chase of the seemingly moving finger but for the limitations enforced by his seating. Yet if rotated and asked to make the same movements of the arm without any finger to touch, he maintained it in the same body-plane with little or no deviation, since he conceived this plane to rotate with his seemingly revolving body. There is, therefore, no cerebellar or automatic drawing of the arm to one side; the past-pointing is purely cerebral.

The authors offer two contributions that so far as they know are new.

"1. The subjective circuit: We postulate this circuit *through the cerebellum* to the higher centers and in proof cite: (a) two patients who had perfect nystagmic reaction but no past-pointing and no vertigo; (b) several cases of spontaneous nystagmus with no vertigo; (c) in most cases of cerebellar lesion dizziness is either absent or subnormal; (d) a patient with no nystagmic reaction and yet normal past-pointing and vertigo. As the two latter reactions seem to go together, it may be concluded, at least for the time being, that the impulses which are con-

cerned with the vertigo pass through the cerebellum. Also, arguing backward, if no vertigo is obtained it may prove that there is a lesion of the cerebellum.

"2. Our second postulate is that the fibers from the different semicircular canals have entirely *separate tracts* of their own; the horizontal canal has an entirely different set from the tracts for the superior canal. This we have proved in twenty-eight cases by means of the following phenomena: In all of these there was little reason to doubt that the region of the posterior longitudinal bundle was implicated. In each of these cases douching of the superior canal (that is with the head upright) produced neither nystagmus, past-pointing, nor vertigo; whereas on stimulation of the horizontal canal (with the head back 90°) all the reactions came through promptly—nystagmus, past-pointing, and vertigo. It is therefore evident that there is a complete central differentiation for the different canals of all three tracts, remembering that in none of these cases was there a peripheral lesion. Autopsy or other findings in some of these cases with no involvement of the horizontal canal fibers of either side, yet with no passing of the stimulus by the superior canal fibers, showed that in the medulla and pons the fibers for the superior canals are mesially located, whereas the horizontal canal tracts are external."

Tests demonstrating neuraxial differentiation of the fibers from the horizontal and the fibers from the vertical semicircular canals are described by Charles K. Mills and Isaac H. Jones.<sup>6</sup>

Cajal has shown histologically that fibers from the vestibular portion of the eighth nerve enter Deiter's nucleus and continue from the inferior cerebellar peduncle into the cerebellum itself. This tract has been generally recognized and accepted. The authors believe that (1) this path includes the fibers from the horizontal semicircular canals exclusively, and (2) the fibers from the vertical canals have an entirely different course. The former are confined to the oblongata, while the latter ascend into the pons.

In thirty-two cases in which the labyrinths themselves and the eighth nerves were normal and the horizontal

(6) Jour. Amer. Med. Assn., Oct. 28, 1916.

canals gave normal reactions, the vertical canals failed in some or all of the well-known responses. That the labyrinths themselves were normal was made probable by the presence of perfect hearing, and corroborative evidence of neuraxial lesions was additional confirmation. In five cases stimulation of the vertical canals produced no nystagmus, no vertigo, no past-pointing and no falling, and yet violent projectile vomiting occurred. This showed that the vertical canals themselves were functioning; in fact, there was even a hyperactive response of the tenth nucleus to the stimulation of the canals. In one case in which the lesion was clearly thrombosis of the right posterior inferior cerebellar artery, the right horizontal canal failed to respond normally, whereas reactions from the right vertical canals were normal.

A study of the data obtained by the ear tests and necropsy in the case reported by them indicates certain definite facts as to the vestibular pathways in the neuraxis. The experiments demonstrated that the tracts from all the semicircular canals except the right verticals, through the vestibular nuclei to the nuclei of the ocular nerves were open. The specimen showed that the Deiters' nucleus group (Deiters' nucleus proper, the triangular nucleus and von Bechterew's nucleus) was unaffected by the lesion, as were also the entire oblongata and the lower part of the pons.

The authors believe that the ear stimulus which produces vertigo passes to the cerebrum through the *cerebellum*. While the paths which carry the vestibular stimuli through the cerebellum to the cerebrum are not demonstrated absolutely in all their extent, the facts at their disposal appear to indicate that they are received by the cerebellum through the inferior cerebellar peduncle from the horizontal canals and the middle cerebellar peduncle from the verticle canals, and after completing their cerebellar itinerary, pass to the cerebrum by way of the superior cerebellar peduncles. In the present case, ear stimulation failed to produce the normal vertigo from all the semicircular canals except the left verticals. Clearly, therefore, there was obstruction somewhere in this vestibulo-cerebello-cerebral pathway concerned with the stimuli causing vertigo. As there

was present a conjugate deviation from the stimulation of the same canals or tracts, the lower neuraxial pathways appeared open. To explain this absence of vertigo the logical place of destruction was at the decussation of the superior cerebellar peduncles. The necropsy studies showed involvement of the superior cerebellar peduncles, which have been demonstrated to contain tracts which pass to the cerebrum from the cerebellum.

With regard to the spontaneous nystagmus upward, this case confirms the previous experience of the writers that such a nystagmus is pathognomonic of a neuraxial lesion, either destructive or due to pressure.

The right vertical canals failed to produce any reactions. It follows, therefore, that the neuraxial fibers from these canals were involved, and as the lesion was in the upper portion of the pons, that these fibers must reach at least as high as the site of this lesion. That the lesion extended further down on the left side suggests that the fibers from the right vertical canals decussate to the opposite side.

In the authors' experience, interruption of the impulses from the vertical canals is produced by tumors of the cerebellopontile angle, internal hydrocephalus causing pressure on the floor of the fourth ventricle, and intracerebellar tumors causing pressure on the pons.

Finally, they are confident that the fibers from the horizontal canal and the fibers from the vertical canals have separate pathways in the neuraxis. That the horizontal canal fibers are confined to the oblongata and enter the cerebellum through the inferior peduncle and that the vertical canal fibers ascend into the pons and enter the cerebellum through the middle peduncle, however, they believe to be highly probable, but feel that the evidence to date is not sufficient to indicate with confidence the exact course of these fibers.

**Localization of Cerebellar Tumors. The Pointing Reaction and the Caloric Test.** Ernest G. Grey<sup>7</sup> presents a paper, the purpose of which is to discuss the importance of the pointing reaction (Barany) and the caloric test in the diagnosis of new growths in the posterior fossa. It is based on an analysis of the

(7) Amer. Jour. Med. Sci., May, 1916.

records of thirty-one cases of cerebellar or extracerebellar tumor.

As Barany, Friesner and Braun, and others have pointed out, in the reaction movements we have a means of differentiating between the balance disturbances due to labyrinthine disease and those of another origin. When the labyrinth is injured, Wilson and Pike and others have shown that the tendency to fall is always in the direction of the slow component of the nystagmus. When the cerebellum is involved, on the other hand, the falling is independent of the direction of the spontaneous nystagmus. The direction of the falling, furthermore, does not change with changes in the position of the head. Barany found that patients with cerebellar disease fall mostly toward the involved side.

In the cases reported, the direction of falling was almost invariably independent of the direction of the nystagmus and the position of the head. It likewise appeared to bear no relation to the side of the lesion. Frequently irrigation of the homolateral ear had no effect on the spontaneous falling, whereas stimulation of the opposite labyrinth resulted in a labyrinthine type of falling.

Neumann, among others, believes that each cerebellar hemisphere exercises control of an inhibitory nature over the nystagmus directed toward its own side. Tumors and other lesions which destroy a hemisphere abolish this inhibitory influence. Accordingly, in such cases, when the labyrinth contralateral to the disease is stimulated with cold water a nystagmus toward the affected side follows which outlasts a normal reaction by several minutes. This so-called enduring nystagmus may last from five to fifteen minutes or more. In the case with hemisphere tumors encountered here no considerable endurance of the nystagmus was noted. Frequently differences were observed in the responses from the two ears, but usually these were due to a diminished reaction on the diseased side. These findings seem to be in accord with the results obtained by Bauer and Leidler from extirpating different parts of the cerebellum in animals. They found that so far as

the cerebellum itself is concerned an increased excitability of the vestibular apparatus only appeared from lesions of the vermis.

Barany and others have found that most of the cases with brain tumor in which there was a considerably increased pressure in the posterior fossa experienced very little subjective discomfort from the caloric tests. The appearance of much dizziness and nausea and of vomiting speaks against a process in the posterior fossa. The results from the irrigations reported here entirely agree with these experiences.

*Conclusions:* The caloric test has proved to be an important means of differentiating labyrinthine from intracranial disease. Together with the pointing reaction, this test has been found of value, furthermore, in localizing circumscribed lesions in the cerebellum. The present report has to do with the results obtained from using these tests in thirty-one patients with cerebellar or extracerebellar tumor.

In most of the patients having cerebellopontine new growths and in certain of those with tumors of one or the other hemisphere, the reactions were sufficiently characteristic to be of supplementary value in localizing the disease. This suggests the desirability of a further study of these tests in neurologic diagnosis. In other patients with intracerebellar or extracerebellar tumor, on the contrary, the results were often ambiguous and afforded no assistance in establishing a diagnosis. In fact, in numerous instances the conclusions drawn from these results were at variance with the other physical findings and, had any great reliance been placed in them, would have led to erroneous results.

There are probably a number of factors responsible for the occurrence of atypical reactions in patients with certified cerebellar tumors. As the more important we may mention first, the great increase in intracranial pressure due to an internal hydrocephalus which ultimately accompanies new growths in the posterior fossa, and second, the diffuse nature of many of the tumors common to the cerebellum.

**The Cranial Nerves.** In a paper based on an analy-

sis of the records of sixty-three cases with intracerebellar or extracerebellar tumors and having to do with the significance of cranial nerve involvement, Ernest G. Grey<sup>8</sup> summarizes his observation as follows:

"Since anosmia in cases with intracranial tumor is usually a distant symptom due to a secondary internal hydrocephalus, it has no appreciable significance for the localization of the new growths. Uncinate gyrus symptoms may appear, secondary to an internal hydrocephalus. The sense of smell was affected in about 7 per cent. of the sixty-three certified cases analyzed in this report.

"While choked disc in itself has no appreciable localizing significance, since it is not infrequently noted comparatively early in the course of certain supratentorial tumors, it may have some importance in this respect when it is associated with other signs. It is our experience that the early appearance and high degree of changes in the eye-grounds, when they appear in company with some of the so-called cerebellar symptoms, are important confirmatory evidence pointing toward a subtentorial localization of the new growth.

"Very little reliance can be placed on an involvement of the third or sixth cranial nerve as a guide to the side occupied by the new growth, in the localization of tumors in one or another part of the posterior fossa.

"The observations recorded here are in favor of the view held by many that the nystagmus seen in cerebellar disease is very frequently of cerebellar origin—an asynergy of the eye muscles. The rule which states that the nystagmus is slower and coarser with the eyes turned toward the tumor is subject to many exceptions. When, however, there is a definite and persisting difference in the size and rate of the jerks with the eyes in the lateral positions, the nystagmus is usually suggestive of the side of the lesion.

"In several patients, when the eyes were directed forward, the slightest change in the position of the eyeballs would bring about a reversal in the direction

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(8) Bull. Johns Hopkins Hosp., September, 1916.

of the nystagmus. In certain others having spontaneous nystagmus with the eyes directed forward, a slight movement to one or the other side of the midline would obliterate the jerks. Fixation—*i. e.*, voluntary effort—as a rule, induced or increased the oscillations of the eyes.

“It was pointed out in an earlier report that there was no discernible relationship between the degree of intracranial pressure and the presence or absence of nystagmus. This finding and the observations recorded above are clinical data in favor of the view held by many that the nystagmus seen in cerebellar disease is very frequently of cerebellar origin—an asynergy of the eye muscles.

“In the paper on nystagmus just referred to, it was shown that this symptom may be absent in certain new growths of the vermis and hemispheres. Caloric tests in most of the cases cited demonstrated an absence of any impairment of the fundamental mechanism of labyrinthine nystagmus. When this report was made, it was suggested that the absence of rhythmic movements of the eyes in a patient exhibiting a cerebellar tumor syndrome points toward an intracerebellar localization of the lesion. Since that time only one case has appeared in this clinic which has proved to be an exception to the rule. In this instance there was a fourth ventricle tumor lying inferior and somewhat posterior to the vermis.

“Since impairments of conjugate deviation of the eyes are only infrequently encountered in the less advanced cases of subtentorial tumor, they have relatively little importance for the localization of tumors within the posterior cranial fossa. Skew deviation of the eyes in cerebellar new growths is rarely seen prior to operation.

“In subtentorial tumors involvements of the fifth cranial nerve have no topographical importance in diagnosis (within the posterior fossa), unless the tumor lies in one or the other cerebellopontine angle. Such a localization is likely only when the homolateral eighth (or seventh) nerve is also affected.

“A paresis or a paralysis of one facial nerve in

tumors of the posterior cranial fossa is strong presumptive evidence of the side of the lesion, though a paresis appears not infrequently in median growths. When the eighth (or the fifth) nerve of the same side is also affected, the diagnosis of a homolateral growth may be made. A questionable involvement of the seventh nerve, on the other hand, is deceptive in this respect due, probably, to the relative frequency of normal facial asymmetries of slight degree.

"In subtentorial new growths a slight unilateral impairment of hearing, which has appeared for the first time in company with general pressure symptoms, is indicative either of a homolateral tumor or, less frequently, of a median growth. When hearing, under similar circumstances, is greatly impaired or lost in one ear, it points toward a homolateral extracerebellar localization of the tumor. Such a diagnosis is confirmed when either the seventh or the fifth nerve of the same side is also affected.

"Tinnitus, it appears, is not a reliable guide to the side occupied by a tumor situated below the tentorium.

"Although vertigo is a prominent symptom of subtentorial tumors as compared with growths situated elsewhere in the brain, it has no appreciable significance for the localization of the disease in one or another part of the posterior fossa.

"The presence of dysarthria and dysphagia (unless they are very marked) in patients with subtentorial tumors, though a source of anxiety, is no contra-indication to operation, since neither is a reliable sign of an impending respiratory paralysis. When they occur, they constitute two of the most striking symptoms of intracerebellar and extracerebellar new growths.

"The spinal accessory nerve is only rarely involved (in less than 5 per cent) in tumors of the posterior cranial fossa. When this nerve is affected, the muscular weakness is not marked and it is homolateral to the growth.

"A weakness of the muscles innervated by one twelfth nerve is of very little significance for the localization of tumors in one or another part of the posterior fossa."

**The Position of the Head and Suboccipital Discomfort.** Of fifty-eight certified cases of cerebellar and extracerebellar tumor examined by Ernest G. Grey,<sup>9</sup> an unusual attitude of the head—tilted so that the ear approximated one shoulder—was found in twenty-three.

In the majority of these the change in position was slight. Of forty-three certified cases of tumor lying anterior to the cerebellum only three showed any tilt of rotation of the head. The unusual attitude in these three cases was scarcely noticeable. These findings indicate that a tilt or rotation of the head in a patient with symptoms pointing toward an intracranial tumor is suggestive of a subtentorial new growth. Backward retraction of the head was a feature in eight of the sixty cases of cerebellar and extracerebellar tumor. Typical opisthotonos attacks appeared in two of these. A similar position was noted in none of the cases with tumors lying anterior to the cerebellum. Some form of suboccipital discomfort was present in forty-four, about 75 per cent. of the cases. Tenderness in the subocciput was found in twenty-one, 36 per cent. Suboccipital headache or pain was complained of in thirty-three, 56 per cent. There was more or less soreness or stiffness of the neck muscles in eighteen, nearly 31 per cent. Of forty-three certified cases with tumors lying anterior to the cerebellum some degree of suboccipital discomfort was found in fourteen, approximately 33 per cent. Suboccipital tenderness was present in eight, nearly 19 per cent. Suboccipital headache or pain appeared in ten, about 23 per cent., soreness or stiffness of the neck muscles in six, 14 per cent. As a rule, occipital discomforts were much less intense in the patients with tumors situated anterior to the cerebellum than in those with subtentorial new growths.

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(9) Ann. Surg., February, 1916.

## DISEASES OF THE SPINAL CORD.

## GENERAL CONSIDERATIONS.

**The Course of the Sensory Tracts in the Spinal Cord.**  
Karl Petren,<sup>1</sup> reviewing ninety-four cases of stab wounds of the spinal cord, places into one group thirty-nine cases showing crossed analgesia and temperature disturbances with no change in touch, the paralysis remaining only on the one side. In another group he places twenty-four cases with one-sided paralysis but with disturbance not only of pain and temperature sense but touch also, and in a third group thirty-one cases in which there was disturbance in the three forms of skin sensation on the crossed side, and in which at first the opposite side was paretic to a greater or lesser degree, but which finally cleared up.

From this it is apparent that the fibers for pain and temperature sense course near to the lateral column of the cord. As to touch, it is immediately seen that a separate course must exist. He thinks that for this sense two courses are present, first in the posterior bundles in the long exogenous fibers, and second in the crossed lateral columns, somewhere along with the pain and temperature sense.

As to muscle sense, he states that in twenty-four cases of Group 1, this sense was normal eleven times, thirteen times affected on the side of the lesion. In ten cases of Group 2 all showed disturbance of the muscle sense on the side of the lesion, and one on both sides.

In fourteen cases of Group 3, nine showed a bilateral disturbance, and five on the side of the lesion. From these findings he concludes that the muscle sense does not run in a crossed tract, that the fibers for muscle sense run medial to the fibers for pain and temperature, because in Group 1, eleven cases were free from disturbances of muscle sense. The fibers for muscle sense cannot run up in the posterior columns alone, because in Group 2, where both posterior columns were affected, only one case showed bilateral disturbance of the

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(1) Neurolog. Centralbl., January, 1916.

muscle sense. Furthermore, the fibers for muscle sense must run lateral to the pyramidal tract because in Group 3, in five cases in which the muscle sense showed disturbance on the side of the lesion only, it was not disturbed on the temporarily paretic opposite side. Therefore, he concludes that the fibers for muscle sense run in two columns, one on the same side in the posterior columns and the other in the lateral cerebellar bundle.

As corroborative evidence he refers to the work of Sanders on Friedreich's ataxia, in which it was found that with severe degenerations of the posterior columns, whereas muscle sense was markedly disturbed, pain, temperature and touch were but little affected, and touch was involved to a greater extent than either pain or temperature sense. This he takes to mean that a double tract exists for the fibers of touch.

**Elective Localization of Bacteria in the Nervous System.** In this paper E. C. Rosenow<sup>2</sup> summarizes briefly the results of an experimental study of the possible etiologic relation of localized foci of infection, especially in and about the teeth and tonsils, to disease of the nervous system. He says that lesions of the spinal cord, usually patchy in character, were observed in 58 per cent. of thirty-one animals injected with the bacteria from the tonsils or infected teeth in three cases of multiple sclerosis. Lesions of the spinal cord, consisting of numerous hemorrhages, especially in the anterior horns in the cervical region, were noted in 78 per cent. of thirty-six animals injected with the staphylococcus from the tonsil in a typical case of sporadic anterior poliomyelitis. Lesions of the meninges and spinal cord occurred in 50 per cent. and 66 per cent. of twenty-one animals injected with the bacteria as isolated from the pyorrheal pockets and tonsils in a case of transverse myelitis with paralysis of the lower extremities. Lesions in or about one or more of the posterior roots occurred in 83 per cent. of eighteen animals following injection of streptococci from cases of brachial, intercostal and post-herpetic neuralgia. The occurrence of neuritis in 28 per cent. of these

(2) Jour. Amer. Med. Ass'n., Aug. 26, 1916.

animals is noteworthy. Lesions of the peripheral nerves occurred in 79 per cent. of the nineteen animals injected with the pneumococcus obtained on two occasions from multiple neuritis. In one rabbit typical wrist-drop developed. Lesions in or about the joints occurred in 48 per cent. of the twenty-nine animals injected, in the myocardium in 28 per cent., and in the muscles in 93 per cent., following injection of the cultures (all containing slightly hemolyzing streptococci) from the twelve cases of "myalgia" (cases of fibrositis or mild myositis, with or without peri-arthritis).

He says:

"The conclusion seems warranted that the lesions in the patients studied were due to hematogenous infection from a focal source by the bacteria isolated. The improvement in symptoms following removal of foci of infection which were proved to harbor bacteria with peculiar localizing powers in multiple sclerosis, transverse myelitis, several cases of persistent neuralgia (although not in others), and in the case of dental neuritis and myositis speaks in favor of this view. The results emphasize the importance of thorough search for and removal, if possible, of foci of infection in diseases of the nervous system. In the light of these results and much clinical evidences, as emphasized especially by Billings, a chronic focus of infection which cannot heal for mechanical reasons, often teeming with bacteria, must be considered as a test tube with a permeable wall embedded in the tissues, which, as I have already jointed out, affords not only abundant opportunity for the entrance of bacteria and their products, but also the conditions favoring the acquisition of various infective powers by the bacteria. The demonstrated presence over a long period of time in foci of infection (particularly in chronic diseases) of bacteria with the same elective localizing powers suggests that the tendency to recurrences of a certain type of disease in the same patient and even hereditary tendencies may be due in part to the peculiar environment furnished by the individual which may favor the acquisition and maintenance by the bacteria of a particular infecting power."

[Although such conclusions may be warranted from Rosenow's findings in cases such as anterior poliomyelitis multiple neuritis, certain cases of myelitis, zoster and myositis, they are far from convincing when applied to such a disease as multiple sclerosis which shows a perfectly pathognomic histologic picture, i. e., destruction of myelin sheaths and preservation of axones.—Ed.]

**Toxi Infection of the Central Nervous System. A Clinical and Experimental Investigation.** David Orr and R. G. Rows,<sup>3</sup> having dealt only with lymphogenous infection of the cord and brain, now contrast the lesions produced by this mechanism with those which occur when a hematogenous intoxication is induced. They undertook a series of experiments in which the abdominal cavity was chosen as the site for infection. This was done for three reasons: (1) The peritoneal cavity is most suitable for an experiment in which one wishes to avoid infection of the lymph system of spinal nerves; (2) to reproduce as closely as possible a gastro-intestinal intoxication, and observe the effects on the spinal cord; (3) to ascertain in how far such toxi-infection affected the sympathetic ganglion chain. Celloidin capsules containing a broth culture of the *Staphylococcus pyogenes aureus* were therefore placed in various regions of the abdomen, where they became attached to the mesentery, kidney, bladder, or lower border of the stomach, etc.

On examining the spinal cord from this series of experiments the authors found no evidence of lymphogenous invasion, either in the sheath of the spinal ganglia, the perineurium of the spinal roots, or in either the dura mater or pia-arachnoid. Within the cord, however, there were very definite changes, which they summarize shortly: (1) The most highly developed structures, the nerve-cells, suffer least of all; (2) there is primary degeneration of the myelin sheath round the cord margin and along the postero-median septum; (3) the myelin degeneration is greatest in the upper part of the cord; (4) there is edema of the cord; (5) there is active proliferation of the perivascular neuroglia; (6) the vessels are dilated, congested, are hyaline, and contain thrombi of

(3) Edinburgh Med. Jour., August, 1916.

the same nature. If these be now contrasted with the cord lesions in lymphogenous infection the difference is at once obvious.

*Lymphogenous* infection is characterized by (1) the reaction of the cells of the fixed connective tissue; (2) the proliferation of the cells of the adventitial sheath of the veins and capillaries; (3) the appearance of numerous scavenger-cells when the myelin is disintegrated; (4) nerve-cell degeneration and neuronophagy. From the above, one must conclude that the lesions in *hematogenous* intoxication differ very widely from those found in lymphogenous infection, where the fixed tissues are actively proliferating and all the morbid phenomena are of an inflammatory type. The difference between the two might, therefore, be expressed by saying that in lymphogenous infection the inflammatory phenomena reach their maximum, in hematogenous intoxication they are reduced to a minimum, and they consider this a most important distinction in neuropathology.

The condition of hyaline thrombosis referred to above varies in degree and in appearance from case to case. Arteries, veins, and capillaries are affected, and the thrombotic change is accompanied by edema, shown by wide dilatation of the perivascular and pericellular spaces.

In incomplete thrombosis the hyaline material lies along each side of the vessel as a thick band, the two sides being connected by trabeculae, thus forming a network in the lumen of the vessel. Frequently, however, the whole lumen is occupied by a homogeneous hyaline mass, or by a structure in which many red corpuscles are clearly evident in various stages of hyaline degeneration.

The leukocytes also undergo hyaline degeneration. Their affinity for acid fuchsin is intensified, they lose their normal shape, become clumped into masses in which are numerous granules which stain deeply with hematoxylin and are at times arranged in a horse-shoe fashion. These granules they regard as remains of degenerate leukocyte nuclei. There are many hyaline threads in the vessel lumen to which the leukocytes frequently contrib-

ute, forming a hyaline syncytium. Purely fibrinous thrombi are not infrequently encountered.

Lesions of practically the same distribution are found in cancer cachexia, pernicious anemia, Addison's disease, etc. It is of importance to note that the gray matter of the cord so richly supplied by the anterior spinal artery may be normal; Goll's tract in the cervical enlargement is attacked in its middle third, leaving the long lumbo-sacral fibers so far away from their trophic center untouched; between the lesions in the cervical, dorsal and lumbar cord there are great differences not to be accounted for by a focal lesion. Such a distribution of degeneration can not be explained by a general intoxication. It fails completely when we consider that the areas supplied by the pial vessels, the cord margin and postero-median septum, show degenerative change, while the regions supplied by the anterior spinal arteries escape. It is highly probable that there must be another factor besides intoxication which determines the localization of the degenerations, and certain indications point to the sympathetic nervous system.

From the above clinical and experimental study it is clear that the two mechanisms of infection of the cerebrospinal system—the hematogenous and lymphogenous—are characterized by sufficiently distinct morbid phenomena, and if we apply the results of the experiments to the human subject we obtain very considerable assistance in arriving at an understanding of the genesis of certain lesions.

A consideration of the phenomena in the subacute non-systemic lesions of the cord, such as occur with or without anemia, Addison's disease, cancer, cachexia, etc., shows that they must be included in the hematogenous category. There is an entire absence of the proliferative change in the adventitia of the veins and capillaries which characterizes the lymphogenous infections. The root entry zones in the posterior columns are, except perhaps in the latest stages of the affection, quite sound, while there is a marked sclerosis around the postero-median septum. The nerve-cells in the gray matter maintain their integrity. The morbid picture is not inflam-

matory in type, and corresponds to what is found in experimental hematogenous intoxication.<sup>3</sup>

#### ACUTE ANTERIOR POLIOMYELITIS.

**Etiology.** E. S. Rosenow, E. B. Towne, and G. W. Wheeler<sup>4</sup> state that they have isolated a peculiar streptococcus from throats, tonsils, abscesses in tonsils and from the central nervous system in cases of poliomyelitis. Paralysis has been produced in animals of various species by intravenous and intracerebral injection of cultures of this organism, and lesions of the gray matter of their nervous system have been demonstrated. From the nervous system of these animals the streptococcus has been isolated in pure culture, while their other tissues were sterile; it is remarkably polymorphous, and appears to grow large or small according to the medium in which it is grown, even after passage through a Berkefeld filter.

Using the organism in its large form, paralysis has been consistently produced in animals known to be insusceptible to inoculation with material from epidemic poliomyelitis as heretofore practiced. After paralysis had been produced in a series of three rabbits, the strain caused characteristic paralysis and lesions of poliomyelitis in monkeys.

The exact relation of the results to the facts already established as to the etiology of poliomyelitis can not yet be definitely stated. It appears to the authors that the small, filterable organism which has been generally accepted as the cause of poliomyelitis may be the form which this streptococcus tends to take under anaërobic conditions in the central nervous system and in suitable culture mediums, while the larger and more typically streptococcic forms, which investigators have considered contaminations, may be the identical organism grown larger under suitable conditions.

(3) The infection of the cord by extension of inflammation from peripheral foci by way of the lymphatics of the nerve trunks was reviewed at some length in the Practical Medicine Series, 1913, Vol. X, p. 104; and further communications were reported in the same Series in 1914, Vol. X, p. 89.

(4) Jour. Amer. Med. Ass'n., Oct. 21, 1916.

**Bacteriologic Observations on Epidemic Poliomyelitis.** George Mathers<sup>5</sup> states that in seven of the eight cases examined so far bacterial growth developed in the aerobic ascites dextrose broth and agar cultures after eighteen hours, while in the anaërobic cultures a definite growth usually did not appear until after from three to seven days, and then often very scantily. In six of the seven instances a pure culture of a Gram-positive micrococcus was obtained. In one instance the cultures gave also a Gram-negative bacillus.

In view of the accepted facts in regard to the virus of epidemic poliomyelitis it would seem most reasonable to regard the micrococcus described as a secondary invader.

**Bacteriologic Findings in Cerebrospinal Fluid in Poliomyelitis.** John W. Nuzum and Maximillian Herzog<sup>6</sup> report the isolation of a Gram-positive micrococcus from the cerebrospinal fluid during life in eight out of nine cases of acute poliomyelitis. These findings have been extended by Nuzum<sup>7</sup> and the same peculiar Gram-positive microorganism has now been isolated from the spinal fluid in forty-five out of fifty cases studied. It presents both the same cultural and morphologic characteristics as does the micrococcus isolated from the brains and spinal cords in fatal human cases at necropsy.

Moreover, the same organism, when injected into monkeys, young lambs and rabbits either intraperitoneally, intravenously or intracerebrally, has produced flaccid paralyzes of the extremities, and in a considerable number of the cases the typical histologic changes in the central nervous system which characterize the disease in man. Paralysis has been readily produced by inoculation of cultures obtained from the human spinal fluid even in the fifth generation. Subcultures two weeks old have produced paralysis of the extremities in the *Macacus rhesus* monkey. The same microorganism injected has been recovered in pure culture in nearly every instance both from the cerebrospinal fluid and from the central nervous system of the paralyzed animals at necropsy. Furthermore, in special stained microscopic sec-

(5) Jour. Amer. Med. Ass'n., Sept. 30, 1916.

(6) Jour. Amer. Med. Ass'n., Oct. 21, 1916.

(7) Jour. Amer. Med. Ass'n., Nov. 11, 1916.

tions of the spinal cord and medulla of these paralyzed animals, this organism is visible in the gray matter, arranged chiefly in pairs but also in short chains.

**The Spinal Fluid in Diagnosis of Poliomyelitis.** Josephine B. Neal and Phebe L. Dubois<sup>8</sup> state that the procedure which they find to be their most reliable and valuable aid in the recognition of poliomyelitis is the examination of the spinal fluid. In poliomyelitis this fluid is usually clear. Very rarely it may be slightly cloudy in the early stages. It often shows a good fibrin web formation. The reduction of Fehling's solution is prompt. There is a slight or moderate increase of albumin and globulin and also of the cellular elements. As a rule, 80 per cent. or more of the cells are mononuclears. In examining such fluids the writers have frequently observed the presence of large mononuclear cells which they believe to be in a measure characteristic of poliomyelitis. Those poliomyelitic fluids which are cloudy present a polymorphonucleosis which may run as high as 90 per cent., but which were usually found to be about 60 per cent.

Two rare types of spinal fluids sometimes occur in poliomyelitis when the hemorrhagic process has been more than usually severe. The first of these is of the true hemorrhagic character, the red blood cells being evenly diffused throughout the field. When collected in successive tubes the specimens are all homogeneous, showing no change in the intensity of the hemorrhage. This serves to differentiate it from bloody fluids obtained by the accidental puncture of a vein.

Evidence of an older hemorrhage occurs in the second of these rarer fluids, which, having a characteristic yellow color and coagulating spontaneously, illustrates the so-called syndrome of Froin. These fluids occur in other conditions, and therefore are not pathognomonic of poliomyelitis.

In early cases of *purulent meningitis* the spinal fluid shows a varying degree of cloudiness, except in very rare instances, when it may be clear. A greater increase in albumin and globulin is usually found here than occurs in poliomyelitis, with a poorer reduction of Fehling's

(8) Amer. Jour. Med. Sci., September, 1916.

solution. The cells in these fluids of purulent meningitis are 90 per cent. or more polymorphonuclears, and the etiologic organism is always found except in the mildest cases. In certain mild cases of meningitis—probably of the epidemic variety—the meningococci may never be positively demonstrated in the fluid. In purulent meningitis due to other organisms these practically always appear later. In one instance only, the authors have seen a clear fluid from an early case of epidemic meningitis of about eighteen hours' standing. Although the cellular reaction was so slight the meningococcus was demonstrated to be present in the fluid by smear and culture.

The fluid in *meningism* is increased in amount, but is practically normal in character.

The fluid in *tuberculous meningitis* most nearly resembles that of poliomyelitis. It is practically always clear with a cellular increase consisting largely of mononuclears, though in very acute cases it may be distinctly cloudy with an excess of polymorphonuclears. The number of cells per cubic centimeter is usually greater than in poliomyelitis; the increase in albumin and globulin is more marked, and the reduction of Fehlings' is not so good.

In rare instances, when clinical signs are confusing and when the results of the cellular examination and chemical analysis are indefinite, and it is impossible to demonstrate the tubercle bacillus in the fluid, a positive diagnosis must wait upon the results of animal inoculation.

Another laboratory method of slight diagnostic value may be mentioned here, the so-called neutralization test. In this, serum from a suspected case in the stage of recovery is mixed with a known fatal dose of an active virus. These are incubated and later injected intracerebrally into monkeys. Failure of the disease to develop indicates that the virus has been neutralized. This test, however, does not furnish conclusive evidence of poliomyelitis, for sera from those known to have been free from a recent attack of the disease have sometimes successfully neutralized the poliomyelitic virus.

**Immunity in Poliomyelitis.** E. W. Taylor<sup>9</sup> says that

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(9) Jour. Nerv. and Ment. Dis., September, 1916.

the question of the possibility of a second attack of poliomyelitis is both of theoretical and practical interest. It is generally recognized and undoubtedly true that a definite immunity results from an attack of the disease. Whether or not this principle is absolutely without exception remains a matter of doubt. Evidence appears to be accumulating, slowly it is true, that the disease may repeat itself in the lifetime of an individual at a varying interval of time, from months to years.

The evidence in the case reported by Taylor points clearly to two distinct attacks of poliomyelitis separated by an interval of three years. In the first attack, the right leg and left arm were chiefly affected, the paralysis of the right leg recovering in great measure, but still showing in some degree at the present time. That this was an actual paralysis at first of a considerable degree of severity is shown, not only by the statement of the mother, who said that the child was obliged to learn to walk over again, taking a year in the process but also more conclusively by a photograph, which showed the child's shoe worn on the inner side of the forward part of the sole, due necessarily to the dragging of the foot in walking, conclusive evidence of the weakness of the anterior group of muscles. The second attack, coming on at the age of 6, affected essentially the left leg, rendering it comparatively useless and making necessary an operation for the relief of the deformity. The diagnosis of poliomyelitis can not be questioned. The onset with fever, followed by a flaccid atrophic paralysis, without sensory involvement and resulting in permanent muscular atrophies, are entirely characteristic and could hardly be due to any other cause. It also does not seem open to dispute that there were in this case two distinct attacks separated by a period of three years of health.

From a survey of the published cases, which it has been possible to collect, it appears that in those reported by Auerbach, Neurath, Lövegren, Sinkler, Friedjung, and Hennelly, the so-called second attack occurred in no instance more than four months after the first.

This interval of time is certainly too short upon which to base a theory of an actual second attack rather than an exacerbation or a relapse of the original infection. It is, however, worthy of note that such relapses should occur, and it certainly can not be definitely asserted that even these are not re-infections. Of the remaining cases, the second attack occurred at the following intervals: Sheppard, sixteen years; Eshner, a somewhat doubtful case inasmuch as the second attack occurred after a fall and without evidence of infection, eleven years; Eckert, six years; Lucas and Osgood, two years and three months; Oulmont and Baudouin, one year (a doubtful case); Sanz, fourteen years; and the case here reported, three years. Lucas and Osgood assume that their patient was a carrier and harbored the virus which again assumed activity, leading to a fresh paralysis, at the end of two years and three months. This, as before, stated, is an assumption which the facts do not necessarily justify. It can not be disproved that an actual second infection occurred. This would seem even more probable in the cases of six, fourteen, and sixteen years respectively, reported by Eckert, Sanz, and Sheppard. This applies also to the case reported in this paper. It is hard to imagine conditions which would permit a virus to remain dormant for these long periods of time, and then suddenly so to increase in virulence that a second paralytic attack is induced, nor does there seem to be an analogy for this in other infectious diseases. A more rational hypothesis, but one which as yet is incapable of experimental proof, is that the first attack in rare cases does not lead to such an immunity that a second and entirely independent attack is impossible. It is at least desirable that all cases in which a second attack is definitely determined should be placed on record.

In general it may be concluded that an attack of poliomyelitis in the majority of cases confers a lasting immunity; that it is definitely established that exacerbations or relapses may occur at short intervals of time after the primary onset; and finally, that evidence is accumulating to show that an actual second

attack with re-infection from an external source may and probably does occur in rare instances.

**Unusual Features of Acute Poliomyelitis.** Included in a lecture by Frederick E. Batten<sup>1</sup> are references to some of the more unusual features of this condition.

*Relapses in poliomyelitis:* Reference was made to the so-called "jump" case of poliomyelitis in which the disease is not steadily progressive, but remains stationary for a day or two and then rapidly advances a further stage; arrest may again take place followed by further advance. Relapsing cases differ from the above in that the patient improves in the interval, and then after some weeks fresh symptoms develop which may again clear up, and then a further relapse may occur.

*Second attacks of poliomyelitis:* These, Batten says, are very rare. The literature contains three or more instances, but none is very convincing, nor will they bear critical investigation.

*Cerebrospinal fluid in poliomyelitis:* The examination of the cerebrospinal fluid in poliomyelitis has yielded variable results. In some cases the fluid has been normal, in others there has been an excess of albumin with a moderate increase of lymphocytes, in others again a very large increase of these cells has been noted. The condition of the cerebrospinal fluid has varied not only with the time after the onset of symptoms at which the fluid was examined, but also with the nature of the case, the cases with the most marked meningeal symptoms tending to yield the largest cell count. Those who have had the opportunity of studying the cerebrospinal fluid in the pre-paralytic stage have noticed a high percentage of polymorphonuclear cells, but in the majority of cases seen in England, when the paralytic signs are well developed, the cell count is always lymphocytic and usually not very high.

*Intra-uterine poliomyelitis:* The occurrence of poliomyelitis in the fetus during intra-uterine life is very difficult to prove. Not a few cases come under observation in which the paralysis is stated to have been observed immediately after birth, and when the child has come

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(1) *Lancet*, April 15, 1916.

under observation the clinical features have been those of poliomyelitis.

*Herpes zoster:* Regarding this disease, Batten says: "In childhood the simultaneous occurrence of herpes and poliomyelitis is rare, and one case is quoted in a boy aged 7 years. Nearly all the recorded cases of paralysis associated with herpes are in adults and in many cases in elderly people, and it seems certain that many of these can not be attributed to the virus of poliomyelitis."

*Serum treatment:* It has been shown experimentally that the serum of patients who have recovered from an attack of poliomyelitis has the power to destroy the virus *in vitro*, but that this immune serum has no power to prevent the development of the disease when injected simultaneously or after the virus has been injected.

*Rest:* There can be no doubt that rest is the most important factor in treatment: the too early movement may in some cases re-start the disease which has become quiescent. A child with an acute attack of poliomyelitis should be kept absolutely at rest in bed for at least three weeks. The principles by which physiologic rest can be secured to the muscles are considered by Batten, and the conclusion at which he arrives is that the paralyzed muscles must be put into the "zero" position, a position defined by Mackenzie as the position in which the individual muscle itself is relaxed and both its own action and that of its opponents prevented. Sherrington has shown that postural contraction which counteracts the effect of gravity can be maintained for long periods without fatigue, and a position which can be maintained without fatigue must be a position of rest.

*Posture:* Various methods have been adopted for keeping the affected muscles at rest. Most of them tend to immobilize the patient for many months. It is important while keeping the completely paralyzed muscles in a normal zero position, and at rest, to encourage other muscles, which may have been more or less affected, to resume their normal function. This is accomplished by re-education. The erect position and walking are the best forms of re-education for the trunk muscles. The method of re-educating the severely paralyzed muscles is described below.

*Splints:* The practical application of the above principles has been carried out by placing the paralyzed limb or limbs in celluloid splints, accurately fitted to the limb as soon as it is possible to make the cast after the onset of the disease.

*Re-education:* In re-educating a severely paralyzed muscle it is important in the early stages to place the limb in such a position that the load on the muscle is at its minimum, and only a very small force is required to overcome the effects of gravity. A bath is an excellent place to practice the re-education of badly paralyzed muscles; for the weight of the limb is supported and ranges of movement are possible which without the support of the water are impossible. Walking exercises and the erect position are the best forms of re-education for the trunk and abdominal muscles. When once the legs are splinted it is possible to get the patient into the erect position, and this allows of the physiologic use of the back, trunk and pelvic muscles. Such walking exercises are carried out by the use of a walking machine which supports the patient both physically and morally; for it is important to give the individual the feeling of complete security.

**Treatment of Acute Poliomyelitis.** S. J. Meltzer<sup>2</sup> arrives at the general conclusion that in cases of poliomyelitis with ascending paralysis, death is due to a respiratory paralysis from an involvement of the origins of the chief respiratory nerves, while in cases of encephalitic poliomyelitis the vasomotor center may be the first vital point which becomes paralyzed, and death is due primarily to a rapid sinking of the blood-pressure.

It is therefore a well-founded assumption that there are some patients with acute poliomyelitis with impending respiratory paralysis, whose life could be saved by application of an efficient method of artificial respiration sustained for some time.

It has been shown that the primary appearance of respiratory paralysis in ascending cases is probably due to the presence of edema and other reversible path-

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(2) New York Med. Jour., Aug. 19, 1916.

ologic phenomena in the corresponding segment of the cord.

It is evident, therefore, that if we could find a means by which the edema and other reversible processes of the peripheral zone could be kept down at will continually, or at least very frequently for long periods, even while the inflammatory process of the primary focus was still active, the nerve tissues subjected to the action of the peripheral zone would derive definite benefit. About thirteen years ago, Meltzer found that adrenaline was such a reliable remedy.

Regarding the use of adrenaline in poliomyelitis he says: "As soon as the diagnosis of poliomyelitis is established, 2 c.c. of 1 in 1,000 adrenaline should be injected every four to six hours. Before the first injection is given, a fairly large quantity of spinal fluid should be withdrawn, the quantity being in proportion to the pressure prevailing in the spinal canal. The subsequent injections should be made without regard to the presence or absence of spinal fluid. Unless the pressure appears to be very high, I advise that in advanced cases not much of spinal fluid be withdrawn, because at this stage the spinal fluid may already contain some useful antibodies. All injections should be washed in with 2 c.c. of the salt solution, but if no spinal fluid is present the adrenaline should be washed in with at least 5 or 6 c.c. of salt solution; in the presence of fluid in the spinal canal the adrenaline will more readily spread all over the cord. In serious cases the quantity of adrenaline administered may be as much as 3 c.c. to each injection. In cases in which the encephalitic symptoms are predominant, however, greater care should be exercised with reference to the quantity of the injected adrenaline; it should be used in inverse proportion to the exciting effect which the injections may produce. In previous experiments I found that adrenaline is often destroyed when mixed with human spinal fluid. I prefer therefore that the injections should be given oftener, at least every four hours when possible. The injections should be continued until from four to five days after all paralysis has disappeared, or at least until no fur-

ther reduction in the extent of the paralysis has taken place."

**Immune Human Serum in the Treatment of Acute Poliomyelitis.** C. W. Wells<sup>4</sup> summarizes his results as follows:

1. The administration of immune serum in acute poliomyelitis is based on recognized principles of immunity.

2. Because the lesions are not confined to the nervous system, and because the lesions therein consist essentially of perivascular infiltration, intravenous injection of serum appears to be a rational procedure, either alone or in combination with intraspinal injection.

3. Intravenous injections of serum should if possible consist of doses of from 50 to 100 c.c. or more daily.

4. Following intravenous or intramuscular injections of serum, spinal fluid should be withdrawn.

5. Intraspinal injection of serum usually produces an increase in the number of the leukocytes with increase in the proportion of polymorphonuclear cells in the spinal fluid.

6. No ill effects have followed the use of serum in this series, either by intravenous or intraspinal injection.

7. In all cases after intravenous injection, and to a less degree after intraspinal injection, a noticeable improvement usually occurred, which, unfortunately, however, in some cases was only transient.

8. Early administration of the serum is urged, necessitating therefore an early diagnosis of the disease; in severe cases late administration of the serum has produced little if any noticeable influence on the course.

#### SPINA BIFIDA.

**Congenital and Acquired Enuresis from Spinal Lesion. A Myelodysplasia. Stretching of the Cauda Equina.** William G. Spiller<sup>5</sup> reviews the literature of

(4) Jour. Amer. Med. Ass'n., Oct. 21, 1916.

(5) Amer. Jour. Med. Sci., April 16, 1916.

myelodysplasia which was described by Alfred Fuchs in 1909 as consisting of anomalies of development and enuresis nocturna, associated often with spina bifida occulta, and depending on imperfect development of the lower part of the cord. The important features of this condition are:

1. Weakness of the sphincters and especially enuresis nocturna persisting after puberty.

2. Syndactylism between the second and third toes, more rarely between the second, third, and fourth toes, still more rarely between the other toes; usually bilateral.

3. Disturbances of sensation, chiefly of temperature sensation, not strictly radicular in type, especially in the feet and more frequently only in the toes.

4. Defect of the sacral canal recognized by the Roentgen-rays.

5. Anomalies of cutaneous and tendon reflexes in the abdomen and lower limbs.

6. Defects in the feet in many cases (pes planus, varus, valgus), sometimes with peroneus weakness, also trophic and vasomotor disturbances in the toes.

Other anomalies that should be sought for are hypertrichosis of the sacral region, lipoma in the coccygeal region, asymmetry of the rima ani, fovea coccygea, or fistula-like depression of the sacrococcygeal region.

He abstracts three cases recently published by C. Bonorino, V Daondo and Mariano R. Cortex.

In all three cases the findings were: Nocturnal enuresis, skeletal malformations, infantilism, ogival palate, cephalofacial hypertrichosis, syndactylism, and sensory disturbances in the big toes.

Whether defective development of the lower parts of the cord without defect in the sacrum and lumbar vertebrae is a common cause of enuresis nocturna with or without the described anomalies of development can not be determined without necropsies, and as yet sufficient attention has not been directed to this subject to enable us to form positive conclusions.

He reports two original cases. One a boy of 18 with pronounced spina bifida with enuresis and flexion of the toes of the left foot. The left leg was weaker and

smaller than the right. The patellar reflex was a little diminished on each side, and the Achilles reflexes were lost. A spina bifida was observed over the sacrum. The other occurred in a boy of 14 who had always had difficulty in retaining urine. After a three-mile bicycle ride weakness in both legs became pronounced, but gradually improved. Four months later the patellar reflexes were diminished and the Achilles reflexes lost. Pain sensation was impaired along the outer edge of the sole of the left foot and on the plantar surface of the toes. There was bilateral foot drop.

Although the back in the lumbar and sacral regions appeared to be entirely normal to sight and palpation, except that slight tenderness could be elicited by deep pressure over the sacrum, the Roentgen rays showed a grave defect of the sacrum and lower lumbar vertebrae.

Spiller states that disturbance of micturition may be acquired by stretching of the lower sacral in bending the trunk forward on the lower limbs. In such a position the lower limbs are severely stretched, and although the bending of the spinal column may be chiefly in the midthoracic region, the lower roots of the spinal cord are likely to be more affected than any others. These roots are the longest and the stretching is more severe in them. An interesting example of this accident is shown in the report of the following case:

J. C., aged 46, was referred to the University Hospital, Oct. 12, 1915, by Dr. J. R. Thompson, of Pittston. About nine or ten years previously while working in the shaft of a mine and stooping over, an elevator descended upon him and he was caught beneath it. He sustained severe pressure in such a manner that his trunk was bent forward on the lower limbs and his sight was affected for a time, but he was not made unconscious. He was taken home and kept on a lounge for several days, but was not paralyzed in the lower limbs. He returned to the mine about a week after the accident, walking with the aid of a stick.

Difficulty in retaining the urine and feces began soon after the accident and persisted about a year. He had incontinence of urine at night at times, but now

has no difficulty in retaining the urine unless he has taken cold. He gets up once or twice at night to urinate, and during the day he voids urine more frequently. He has no loss of sexual desire, but power of erection has been lost, and he has no seminal emissions. The Achilles reflexes are lost, the right patellar reflex is also impaired, and the left patellar reflex seems a little exaggerated. He has some indication of Romberg's sign. Pain and tactile sensations about the anus and in the perineum are normal, as in the lower limbs.

**A Bed-Wetting Family and Hereditary Spina Bifida.**

D. Jancke<sup>6</sup> reports a family in which the paternal grandfather wet his pants, the father had a weak bladder, and the oldest son, the patient, 26 years old, has never been able to retain his urine. He showed a defect of the sacrum. The second son is healthy, the third often wets himself. The patient has a daughter who wets her dress daily, a boy who wets his clothes and bed and who has a sacral defect, and another daughter who is always wet.

**INJURIES OF THE SPINAL CORD.**

**Gunshot Wounds and Injuries of the Spinal Cord.**

As the results of a hitherto unparalleled opportunity to investigate the physiology, pathology and symptomatology of the spinal cord, James Collier<sup>8</sup> invites attention to the following subjects. He roughly classifies the lesions as follows:

"1. Direct lesions. I would define as a direct lesion any lesion resulting from the passage of a missile across the spinal canal whether it touch the spinal cord or not.

"2. Indirect lesions: (a) Those due to the in-driving of bone, etc., into the spinal canal. (b) Impact lesions where the missile strikes against the bony wall of the spinal canal. (c) Concussion lesions from the shock of high explosions.

"3. Secondary lesions: perithecal and intrathecal

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(6) Deutsch. Zeitschr. f. Nervenheilk., December, 1915.

(8) Lancet, April, 1916.

hemorrhage, medullary hemorrhage and thrombosis, meningitis, edema. These lesions are important as the cause of the deepening of symptoms often at a considerable time after the injury.

"4. Remote lesions which may be found anywhere in the spinal cord and chiefly near the surface: spots of necrosis, sieve-like rarefaction, punctiform hemorrhages, edema, swelling of axons. These lesions are produced by the sudden raising of the intraspinal pressure caused by the passage of the missiles through the intraspinal space or by a general concussion effect. They are well-marked in cases of concussion without external wound."

Of reflex action he says:

"There may be four consecutive stages in the condition of the plantar reflexes following a transverse lesion of the cord: 1. An initial extensor response. 2. Either a complete absence of any reflex, which may be the result of shock or of isolation alteration, or a reduced flexion reflex which is the result of isolation alteration which may come on rapidly. 3. The extensor response which when persistent is indicative of a less severe lesion or alternatively of more recovery than the reduced flexion reflex. 4. The normal flexion reflex which returns when recovery is complete. I have repeatedly observed the successive changes from no reflex to the reduced flexion response, and from this to the extensor response in cases in which some improvement is occurring; and conversely I have seen the extensor response change to the reduced flexion response and the subsequent loss of all reflex action in severe cases which were not improving, and in which the only attributable cause for such a change seemed to be the long-standing isolation of the distal segment of the cord. The condition of the plantar reflex is therefore an index of the severity of the damage to the spinal cord and an important early indication as to whether recovery is occurring or not."

Another most important point which he states that the experience of this war has brought out is that the influence of the higher nervous system on the reflex action and muscle tone of the spinal cord is strictly homolateral so far as the cord is concerned. That is to say,

a total unilateral lesion of the cord produces complete flaccidity with loss of knee-jerk and ankle-jerk and reduction of superficial reflex on the side of the lesion. This will explain those cases in which we find an extensor response upon one side and a reduced flexor response upon the other, in that one has a transverse lesion more complete upon one side than upon the other.

*Contracture* is a phenomenon which is intimately associated with the condition of reflex action. In these paraplegic cases under consideration there are three conditions of contracture of the feet: 1. The dropped foot with retracted toes. This is the ordinary pes cavus of spastic states—the crytallization of the extensor response. 2. The retracted foot with retracted toes. The calcaneus position is often extreme. The ankle-jerk is always lost and the anterior tibial-jerk marked. An extensor plantar reflex is always present. 3. The dropped foot with dropped toes, the position being similar to that of peripheral neuritis. The plantar reflex is either absent or there is a reduced flexor response.

Collier has observed in several cases the dropped foot with dropped toes gradually change into the spastic pes cavus with a change of the plantar reflex from the reduced flexor to the extensor type in cases where some improvement has taken place. Therefore he considers that the dropped foot with dropped toes indicates a more complete transverse lesion and that it is produced by a relatively greater muscular tone in the flexors of the toes than elsewhere, and that it is associated with the reduced flexor response just as the pes cavus is associated with the extensor response in less severe transverse lesions.

The calcaneus position with retracted toes is a very remarkable phenomenon. It has been persistent in those cases in which the author has met it, and has been always associated with loss of knee- and ankle-jerk and increase of anterior tibial and hamstring jerks. The extensor response and active withdrawal reflex have always been present.

As to sensation, he says that there is no crossing of the sensory fibers in the cord below the last dorsal

segment. The path for painful and thermal stimuli crosses more rapidly than does touch. Consequently a unilateral lesion of the cord does not produce a Brown-Séquard syndrome when the lesion is at the level of the first lumbar segment or below this.

The symptoms of the unilateral lesion are as follows: Motor paralysis, loss of sense of position and of passive movement and of appreciation of vibration, form, and compasses upon the same side below the lesion, and loss of pain, heat, and cold upon the opposite side. The bilateral loss in the region of the lesion from the involvement of both right and left crossing fibers is narrower for pain and temperature than for touch. This bilateral loss is much narrower in the dorsal than in the cervical regions on account of the increasing obliquity of the crossing fibers. In the majority of his cases of unilateral lesion of the cord the vibration sense has been lost or most affected upon the opposite side, and this has obtained both in severe and in slight lesions. He admits that he has no pathologic evidence in such a case.

Remote pains are very obstinate in some cases.

To the usual types of paraplegia resulting from lesions of the spinal cord and representing different degrees of isolation of the distal segment—namely, “paralysis in extension” and “paralysis in flexion”—Collier thinks that we might speak of a third type—that of complete flaccid paralysis. For it is from this stage of complete flaccid paralysis that the other types successively develop when improvement is occurring, and to this type they successively return when there is an increasing lesion or from long-standing isolation of the distal segment. There is, of course, no sharp separation between these types; they merge into one another gradually.

In differentiation between root lesions and central lesions the following points may be useful in the diagnosis:

1. In the cervical region extensive root lesions are only encountered when there is severe injury of the bones, especially of the transverse processes and spines, and these are recognizable by deformity, swelling and

edema, and radiography in the absence of these signs; an atrophic palsy of the arms is probably the result of a central lesion, if it is extensive.

2. In central lesions the upper limit of the sensory loss is a line more or less transverse to the axis of the limb—that is to say, the sensory loss is of the “glove” or “stocking” pattern, in contrast to the more or less longitudinal limitation which obtains in root lesions.

3. In the cervical region a relative escape of the long columns or early signs of recovery in these with severe paralysis of the upper limbs is in favor of a root lesion, and may suggest the correct diagnosis at an early stage.

4. In the lumbo-sacral region the most certain indication is the level of the wound of the spine if this can be determined with certainty.

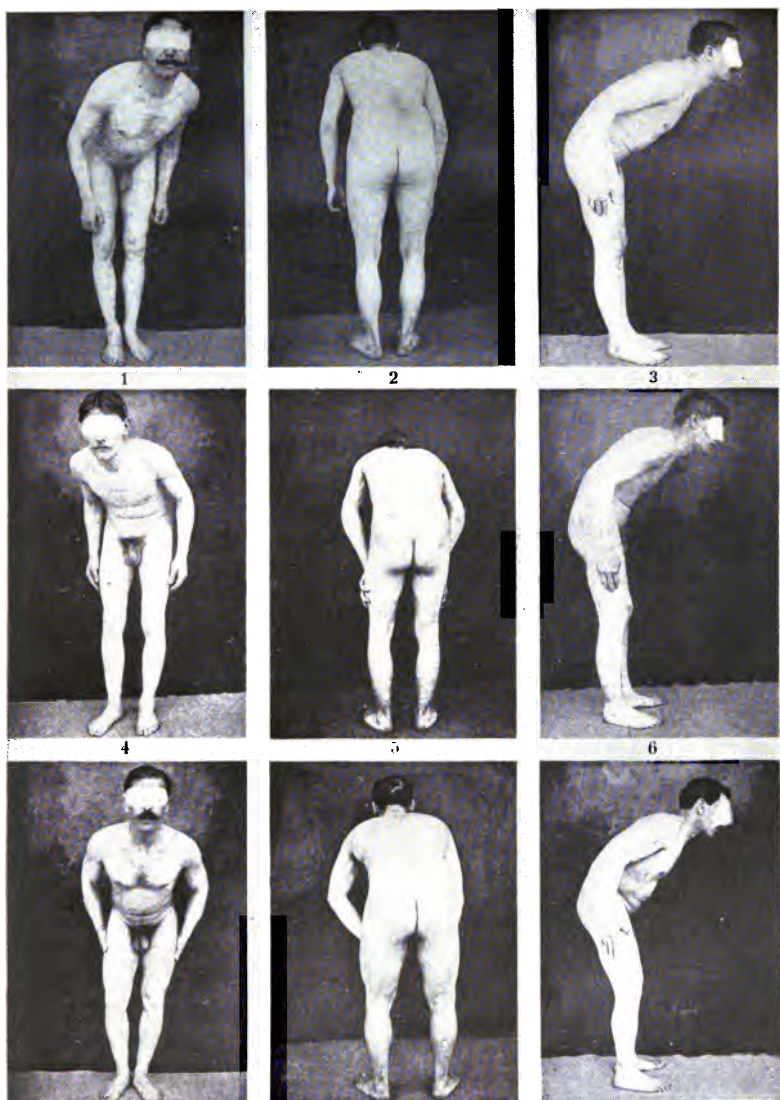
As to prognosis, he states that the early re-appearance of the plantar reflex if lost at first and the change from the flexor to the extensor type soon after the seventh day, or the presence of an extensor response earlier than this, are certain indications that the lesion is partial and that some recovery will occur. Persistent loss of the plantar reflex and long-lasting flexor response are indications that the lesion is severe and that useful recovery is highly improbable. Early return of the knee-jerk and of the ankle-jerk is of good prognostic import. Even in the less severe cases the knee- and ankle-jerks do not return before the fourth day, and their re-appearance before this date gives hope that complete recovery may occur.

**War Injuries of the Spine.** These papers form the Goulstonian Lectures for the year 1915. Gordon Holmes<sup>o</sup> deals in the first lecture with the pathology of acute spinal injuries. He classifies the injuries of the cord into direct injury, contusion and concussion.

On examining a case in which the spinal cord has been completely divided, we find as a rule a considerable amount of clot and often pieces of bone between its two ends. A few days after the infliction of the wound these are swollen, irregular and very soft to touch for at least 1 cm. from the point of division; indeed they may be more or less diffuent.

(9) Brit. Med. Jour., Nov. 27, Dec. 4, 11, 1915.

PLATE VII.



Camptocormie.—Rosanoff-Saloff (see page 147).



Microscopic examination always shows that there are severe and relatively extensive changes in the spinal cord immediately above and below the lesion; for the distance of half a segment at least and often further the tissue is completely softened and none of its normal elements are recognizable. Under the microscope, only ruptured disintegrating axis cylinders and globules and irregular masses of myeline can be seen; the latter rapidly become smaller and smaller and are absorbed by the numerous scavenger cells which quickly pervade the tissue. Hemorrhages, generally small but occasionally of considerable size, are usually found in these softened areas, but they are more common and, as a rule, larger in the gray than in the white matter.

The most striking feature in these sections, however, is the presence of large spherical or irregular cells, which are frequently, apart from the blood-vessels, the only recognizable tissue elements.

These are obviously compound granule cells, the origin of which has been much discussed; by some they are attributed to connective tissue, endothelial or leukocytic origin, but others believe they are wholly, or in greater part at least, derived from the neuroglia.

Two types of change are seen in the nerve cells of the gray matter: in the region of the wound they are often shrunk, stain darkly, and present none of the normal details, or if the tissue is edematous they may be swollen. Some of them at least break up rapidly, often owing to invasion by neurophages, but when severe these changes are evidently recoverable. A more common type of change is chromatolysis, with some swelling of the cell body, eccentricity of the nucleus and disappearance of the Nissl bodies, especially from its center. Beilschowsky preparations often show irregular swelling of the dendrites of these cells.

Occasionally there are small circumscribed patches of necrosis in which both the fibers and the neuroglial matrix are involved, or in which only the larger strands of neuroglia persist, without any evidence of hemorrhage or inflammatory reaction around them.

There is swelling of the axis cylinders. Frequently all those in a considerable area of the white matter are

found slightly swollen in the neighborhood of the wound where the tissue is edematous, but a more unexpected condition is the relatively enormous increase in size of either isolated axones or groups of axones, unassociated with any other obvious changes in the tissue.

Whatever may be the exact mechanism of spinal concussion, it must be admitted that a sudden violent impact on the vertebral column can produce diffuse, irregular and severe structural changes within the spinal cord. The factors which determine the severity of these lesions must be the momentum of the projectile, the part and surface area of the vertebra which it strikes, and the region of the spine which is wounded. He concludes this first lecture as follows:

1. The structural lesions in the spinal injuries of warfare are rarely sharply limited or circumscribed, and can not be compared to those produced experimentally in a physiologic laboratory. The level of the lesion, as indicated by the clinical symptoms, for instance, often does not correspond with the level of maximal damage.

2. The lesions are so irregular in distribution and severity when the spinal injury is not complete that much care is necessary in drawing conclusions from the clinical symptoms alone on the function of parts which it may be assumed have been involved.

3. Secondary changes may occur later in the cord which can alter or modify the clinical symptoms.

The second lecture deals with the clinical symptoms of gunshot injuries of the spine. In studying this phase over 300 cases were observed. The segmental level of the lesion usually can be recognized as accurately by the extent of the motor paralysis as by the upper border of the sensory disturbance. In incomplete lesions, especially if unilateral, an exact diagnosis may not always be possible from even an accurate sensory chart on account of the oblique course of the decussating sensory fibers in the cord. In lesions of the dorsal columns the disturbance in the perception of vibrations is valuable, as it is not recognized below the corresponding segmental area. The view that in total transverse lesions of the cord the reflexes are permanently abolished was confirmed. In less severe lesions the knee-jerks return within

two or three weeks. This absence appears to be due to "spinal shock;" *i. e.*, the sudden severing of a portion of the cord. Spinal shock exhibits itself only in segments distal to the lesions. The reflex over-activity of isolated segments, however, may show itself in automatic movements. An incomplete lesion of the cervical enlargement may seriously paralyze the arm and leave the power of movement in the lower limbs unaffected. Lesion from the second cervical to the second dorsal may produce disturbance in the function of the cervical sympathetic, necrosis pseudo-ptosis, etc. Injury to the lower part of the cervical enlargement was characterized by subnormal temperature, slow pulse, low blood-pressure, scarcity secretion of urine and stupor or lethargy. The condition strongly resembles that of an animal in hibernation. Lesions of the upper cervical segments were sometimes associated with hyperpyrexia. Lesions of the mid dorsal region were associated at times with persistent vomiting. Herpes was present in nine cases above or at the upper margin of sensory loss.

In the third lecture, "The Sensory Disturbance in Spinal Injury," Holmes formulates the conclusions that pain and temperature sense pass through the opposite lateral column, the muscle sense through the homolateral dorsal column and both paths are open to touch.

In the mid dorsal region crossing of pain and thermal impulses occur quickly, is complete in about one segment. In the upper dorsal segments two are required, while higher up in the fourth cervical the decussation of pain sense is complete only in five to six, and the thermal in four to five.

**Post-Traumatic Fixed Flexion of Spine; "Camptocormia."** In February, 1915, A. Souques<sup>1</sup> presented to the Paris Neurological Society four soldiers afflicted with forward curvature of the spine, described as "neuropathic pseudo-contracture." At a subsequent meeting Souques and Mme. Rosanoff-Saloff<sup>2</sup> proposed the term "Camptocormia" (formed from words meaning bending of trunk) for the condition which they consider worthy of a special designation. Still later,

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(1) Rev. Neurol., May-June, 1915.

(2) Rev. Neurol., November-December, 1915.

Mme. Rosanoff-Saloff\* has produced a profusely illustrated and detailed article on the subject.

This form of curvature, or "incurvature" as Souques prefers to call it, has also been commented on by J. A. Sicard<sup>4</sup> who describes four types of "spondylitis" following shell shock: (1) spondylitis with kyphosis; (2) spondylitis with kyphoscoliosis; (3) spondylitis with lumbar rigidity; (4) spondylitis with total rigidity without curvature. The condition may be organic or hysteric, much more frequently the former, according to Sicard, who made the interesting observation that in fully three-fourths of the cases there is marked increase in albuminous substances in the spinal fluid without increase in cells. This albuminous increase is considered due to edema produced by vertebral compression sufficient to interfere with venous circulation.

However, Mme. Rosanoff-Saloff, on the basis of sixteen cases, assures us that "Camptocormia" is a functional condition and essentially similar to normal bending forward of the body (Plates VII-VIII.) except that the head is kept extended for the purpose of enlarging the field of vision. Permanent transverse grooves develop on the abdomen. Except in the early stage, walking is little interfered with and the patients can readily pick up objects from the ground. But they can not straighten themselves in the upright position and the attempt may cause prolonged tremor in the legs. On the other hand, when lying down most patients can readily straighten and even hyper-extend the back. There is no tenderness on pressure on the spinous processes but the lumbar muscles are usually tender. Sensation, reflexes and x-ray appearances of the back are normal. Examination of the spinal fluid made from eight to ten months after the onset showed nothing abnormal.

Nearly all of these patients were merely the victims of "shell-shock" and not actually wounded though often they had been covered by earth or knocked over

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(3) Nouvelle Inconogr. de la Salpetriere 1916, vol. 28, p. 28.

(4) Bull. X, Mem. de la soc. méd. des hôp. de Paris, July 9, 1915.

on the back, with more or less loss of consciousness. Severe pain in the lumbar regions is the earliest and most persistent symptom. The favorite position of the patient in the early stages is to sit "with the head bent forward between the legs." Any movement or change in position causes severe pain and after two or three weeks when the pain becomes less intense the patient tries to stand up straight and finds he can not.

As to pathogenesis, the author believes that during the first weeks of pain and immobilization the mind becomes fixed on the flexed attitude and as all of these persons are neuropaths—a very important etiologic point—the attitude becomes a fixed one.

A successful method of treatment was hit on by Souques, who devised a form of corset which was applied with the patient lying down with the back straight. Those who could not straighten the back even when lying down were anesthetized. While wearing the corset the patients were subjected to strict discipline, forbidden to have visitors or receive letters, so as to reinforce their desire to get along without the corset.

## DISEASES OF THE PERIPHERAL NERVES.

**Recurrent Paralysis of the Facial Nerve, and Its Relation to the So-Called Facioplegic Migraine.** The paralytic complications of migraine, especially those of the cranial nerves constitute an interesting and important chapter in the symptomatology of this disease. Curiously the motor cranial nerves other than the third, fourth and sixth, are not affected with the exception of the seventh, the so-called facioplegic migraine.

J. Ramsay Hunt<sup>1</sup> reviewing and analyzing the cases reported, and adding the cases of recurrent facial palsy, accompanied by pain, remarks that the recurrent or relapsing facial palsy associated with pain in the ear and occipital region is, therefore, merely a peripheral paralysis of the seventh nerve, in which is manifested a peculiar tendency to multiple attacks or recurrences. The symptomatology corresponds in all its essentials

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(1) Jour. Amer. Med. Ass'n., March 18, 1916.

to the more usual type in which there is but a single attack, and similar etiologic factors are also in evidence. Some emphasis may be placed on the theory of a narrow exit at the stylomastoid foramen, which was advanced by Despaigne in explanation of these recurrences, and which might predispose the nerve to compression from very slight inflammatory cause. Such an anomaly might well be inherited. This, however, is only an ingenious theory, and calls for more definite pathologic confirmation.

Facial palsy as a sequel of the migraine attack, the *facioplegic migraine* of some writers, is not a clinical entity. At the present time there are no adequate reasons for the acceptance of such a clinical type. The Rossolimo case which forms the chief support of this teaching is evidently only a recurrent facial palsy with marked sensory symptoms in a woman afflicted with migraine, and one searches the literature in vain for examples of a true facioplegic migraine. Furthermore the fragmentary case report of Hatchek, which is sometimes spoken of as a periodic facial palsy, as this term was used by Moebius to describe the periodical oculomotor palsies, is an equally erroneous interpretation. The relapses in the Hatchek case, conservatively interpreted, represent nothing more than pressure or traction of the facial nerve, this giving rise to intermittence in the paralysis, an occurrence which is by no means rare as a forerunner of permanent palsy in cases of subtentorial tumors. If the facial nerve has any relation to migraine, which is so well established in the case of the ocular nerves, this relationship has yet to be demonstrated. The cases thus far cited do not furnish sufficient ground for any such assumption. Therefore titles like "periodic facial palsy" and "facioplegic migraine" are misnomers which have crept into some of the best monographs dealing with this subject. Such terms are misleading and denote nothing more than transient intermittent facial palsy as a focal symptom of basal tumor in the one case, and the not uncommon relapsing facial palsy associated with pain in the other. It is of course self-evident, says Hunt, that migraine and facial palsy, both of which are

common affections, may be encountered in the same individual but are etiologically distinct.

**Facial Diplegia in Multiple Neuritis.** Hugh T. Patrick<sup>2</sup> presents a paper, saying that in 1908 Laurans collected nineteen cases of facial diplegia in multiple neuritis. To this list Patrick has added twenty-nine, including his own, but perhaps two or three of these might be considered doubtful besides half a dozen that he had taken at second hand, the original reports not being accessible. Perhaps the most frequent type was a not very severe multiple neuritis beginning in the legs, affecting the arms less, with total facial diplegia. The combination of complete double facial palsy with less intense involvement of the extremities was the striking feature. In only one rare instance was the facial palsy partial. But the degree of quadriplegia was exceedingly variable.

As one would expect, sometimes the legs are badly affected and the arms escape, but the arms may be weaker than the legs. Following the rule, the distal muscles are weakest and slower to recover, but occasionally the pelvic girdle or shoulder girdle suffers most. In at least seven cases other cranial nerves were involved, but as a rule for a short time only. Optic neuritis has been observed, once involvement of the auditory nerve.

Assuming that the multiple neuritis is always caused by a poison (or infection), and knowing that certain tissues are peculiarly vulnerable to certain poisons, (wrist-drop from lead, foot-drop from arsenic, paralysis of palate and ciliary muscle from diphtheria), one naturally asks whether the facial palsy may not be due to a particular toxic agent. This can not confidently be answered in the affirmative. But the negative aspect is interesting. Apparently the most frequent causes of multiple neuritis do not cause facial diplegia. No case has been due to a metallic poison (except probably one of plumbism), and Patrick believes no case due to alcohol, although one patient was known to be alcoholic. He has found no typical case due to diphtheria, though of 171 cases of diphtheritic

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(2) Jour. Nerv. and Ment. Dis., October, 1916.

paralysis collected by Ross, five showed more or less facial diplegia, and of fifty cases of precocious palatal paralysis in diphtheria collected by Rolleston two had labial palsy. There is no case following typhoid unless Patrick's first case be one. The most frequently surmised cause has been influenza but in no case was this definitely ascertained and in only four out of the thirty-four cases was it thought highly probable. One case, followed, not immediately, a non-suppurative lymphangitis of the hand and arm, four a longer or shorter period after a throat or tonsillar infection, but in no instance was a culture made. One case occurred in the course of gonorrhea and one following confinement, though there is said to have been no puerperal infection. At least one may have been due to ptomaine poisoning and one occurred in what was supposed to be malarial polyneuritis. But in most cases the signs of infection were vague and not severe and in a good many there was no prodromal illness at all. In a very few the constitutional signs were severe.

He reports a case of facial diplegia occurring in a woman of 21 who developed multiple neuritis as the result of a condition which manifested itself by a fever of three weeks. The temperature fluctuated much as in typhoid but, aside from a few doubtful rose-spots on the abdomen, other symptoms of typhoid were lacking. The Widal test was negative. This fever occurred during a rather prevalent epidemic of what was currently called "grippe" but the organism found was generally a streptococcus or the *Micrococcus catarrhalis* (Plate IX.).

**Neuritis of Cochlear Division of the Eight Nerves Following Exposure to Cold.** J. S. Fraser<sup>3</sup> states that cold in the form of a cold wind or draught blowing on one ear may give rise to a lesion of the auditory or the facial nerve (or both). He reports a case in a woman aged 30, who was seen on Dec. 7, 1914. The patient stated that a week before she had been exposed to severe cold, just at the onset of her menstrual period. Her ears had always been very sensitive, and she had usually guarded them from cold wind by

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(3) Edinburgh Med. Jour., January, 1916.

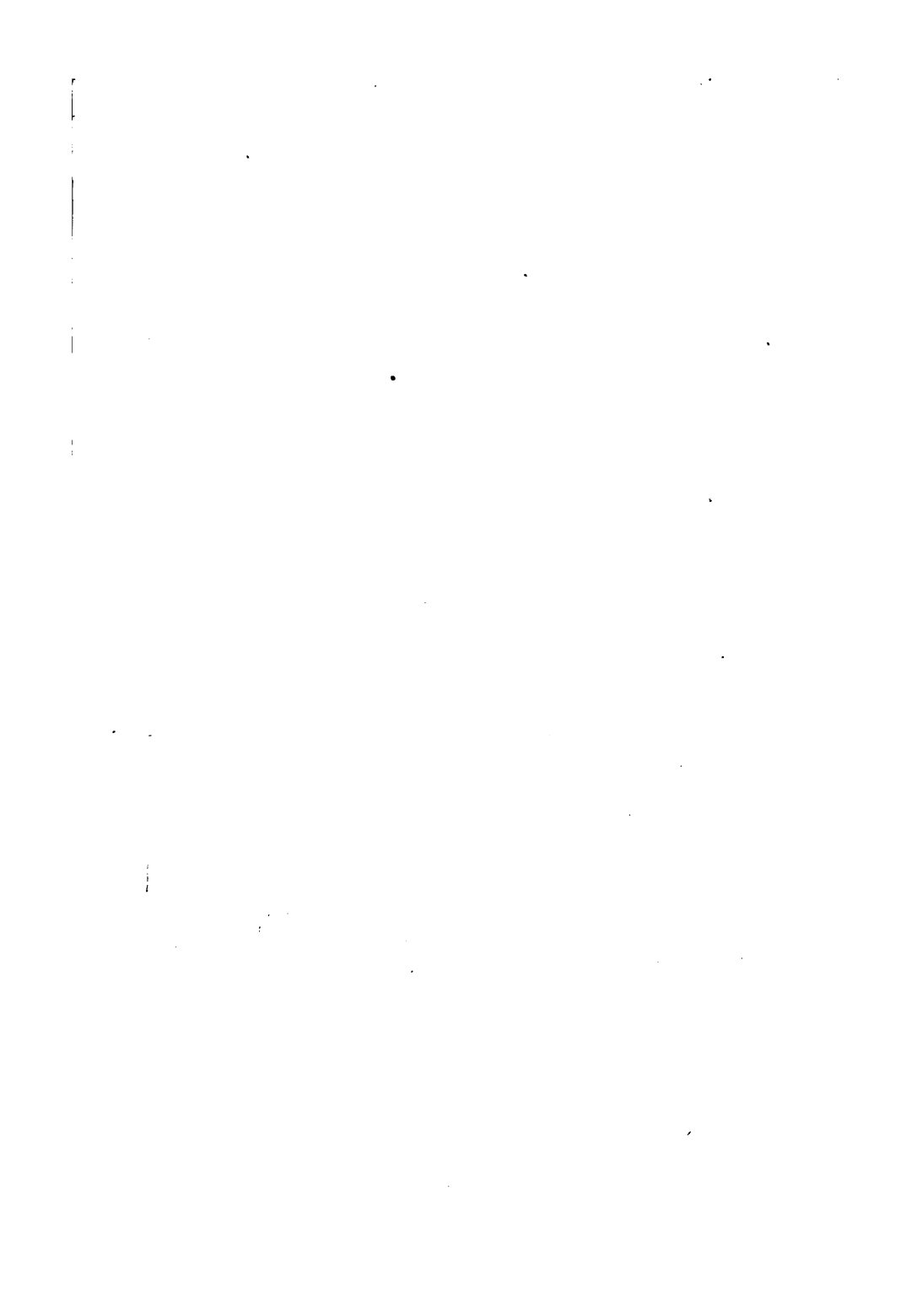


PLATE IX.



Case of facial diplegia in multiple neuritis. Appearance of patient when she tried to close the eyes tightly and spread the mouth as in showing the teeth. Note inability to close the eyes and entire absence of contraction of lower facial muscles.—Patrick (see page 151).

holding her muff up to her face. On waking up in the morning after the exposure to cold she complained of deafness, and noises in the left ear like the escape of gas. There was no giddiness or facial paralysis.

*Examination:* Examination of the nose and pharynx showed normal conditions. Both drumheads were healthy. There were no signs of otitis media. The watch was heard on the forehead and both mastoids. By the right ear it was heard at normal distance, but in the left side the patient could not hear the watch even on contact.

*Apparatus:* There was evidently no reduction of sensibility of the vestibular apparatus.

Fraser remarks that Rutti has pointed out that in labyrinthitis both parts of the inner ear are affected because the condition begins as an inflammation in the perilymphatic space—a space which is common both to the cochlear and to the vestibular parts of the inner ear. On the other hand, in neuritis of the eighth nerve one or other of the divisions (cochlear or vestibular) may be affected alone—as in the present case.

**Gunshot Wounds of the Peripheral Nerves.** O. Foerster<sup>4</sup> reports his review of 600 gunshot wounds of the peripheral nerves. Complete section of peripheral nerves occurs more frequently than was formerly supposed, but there also occur lasting paralyses with severe reaction of degeneration when the nerves appear intact and form no adhesions with the surrounding tissue.

Internal neurolysis (separation of nerve fibers after splitting the sheath) makes plain in such cases the cause of injury because the isolated fascicles are compressed by scar tissue atrophied and in part totally severed.

Dissociated motor paralysis, that is, the intactness of certain muscles in lesions of peripheral nerve trunks, depends in part upon differences in vulnerability of nerve fibers for certain muscles, but chiefly it rests on the anatomic fact that only certain fibers within the nerve sheath are cut. Severe paralysis usually is associated with complete reaction of degeneration, but exceptions are present as to the parallelism. At times complete reaction of degeneration may be present, in the

(4) Berlin. klin. Wochenschr., August, 1915.

face of little paresis, whereas electrical reactions may become normal and complete loss of function remain. Following nerve suture, electrical reactions and voluntary movements show no constant relationship. In some cases the voluntary movement returns first while nerve and muscle are invisible to faradism. In other cases this is reversed. Such cases may be found in lesions situated high up in the nerve.

Frequently nerve lesions are complicated by direct muscle injuries and incorporation of tendons of scar tissue. Occasionally we find irritation of motor nerves so that muscles innervated by them are in lasting tonic contraction and show mechanism of hyperirritability.

The sensory disturbances play a large rôle in the symptomatology of nerve injuries. Disturbances of pure sensory nerves are common; these very often are the cause of severe obstinate neurolysis. The results of such lesions are not only pains and hyperesthesias, but contractures caused by centripetal irritation. These always disappear when the irritation is removed. The position of contracture is typical for each sensory nerve involved. In mixed nerves sensory symptoms also play a large part, pains of great severity of strange character, paresthesia and hyperesthesias are predominant in the beginning and later stages. Besides pain, very disagreeable paresthesias are present, severe burning, and a feeling of extreme dryness of the skin so that patients spontaneously keep the involved territory wet. It is remarkable that sensory stimulus which strikes the nervous system leads to severe pain in the disturbed territory. Patients with sensory median irritation or radial or ischiatic irritation, when hearing music or seeing a bright light, or when a perfectly sound part of the body is touched, experience a pain or disagreeable sensation in the distribution of the injured nerve, every normal sensory centripetal stimulus irradiates in the periphery of the diseased nerve territory. Anesthesias are just as common at times, complete other times but very slight.

Just as isolated motor fibers are found affected, so can single sensory fibers be involved, as in ischiatic injuries, the plantar medianis or lateralis or suralis or

rami calcanei may be selected. Sensory symptoms are very common in lesions of mixed nerves. Profuse sweating, extreme dryness of the skin, with scaling, redness, especially blue-red lividity, more seldom anomalous paleness, high grade coldness, abnormal development of pain, brittleness of nails, etc., belong to the secretory, vasomotor and trophic symptoms.

In treatment Foerster recommends great conservatism; when operations are to be performed he advises plastic nerve repair, employing separate pieces of sensory nerves, using from two to four pieces building up the part intervening between the proximal and distal part of the injured nerve to its original thickness.

Obstinate neuralgias due to injuries of sensory nerves and which do not give way to treatment by alcohol and novocaine injections hot air and diathermia and which are associated with contractures indicate surgical interference. The excision of a neuroma followed by nerve rolling at once gives great relief.

**The Dissociation of Cutaneous Sensations in Injuries to Peripheral Nerves.** Summarizing his results, Donald E. Core<sup>5</sup> says: that it is possible in the majority of cases to diagnose the condition of the affected nerve from the dissociation of cutaneous sensations encountered.

In complete division the loss to brush is more extensive than that to needle-point (cutaneous pain).

A dissociation characteristic of complete division associated with one significant of compression is occasionally met with. This leads to a "lateral displacement" of the two areas of sensory loss; it is met with when more than one nerve is injured, and when the nerves involved supply contiguous areas of the skin. It indicates the complete division of one of these and the compression of the other. When such a dissociation is met with in the territory of one nerve it is possible that it is the result of partial division of the nerve with compression of the undivided fibers.

An undulating form of dissociation, where the limiting edges of the two areas of sensory loss approach and recede or constantly cross each other, is met with in cases that

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(5) Lancet, April, 1916.

are at a standstill. No satisfactory explanation has as yet been arrived at for this type.

In certain areas there is no dissociation; the two areas of sensory loss coincide. Core has not found this common except in lesions of the external popliteal nerve.

**Tardy or Late Paralysis of the Ulnar Nerve.** The type of neuritis which is the subject of J. Ramsay Hunt's<sup>6</sup> paper is peculiar, in that it develops many years after fractures and dislocations about the elbow-joint. For this reason it has been termed "tardy" or "late" paralysis. Usually, the injury to the joint has been received in childhood, the first symptoms of ulnar neuritis making their appearance in adult life. Thus, an interval which may vary from six to thirty-five years elapses between the initial injury and the first symptoms of ulnar neuritis, which are then gradually progressive.

This remarkably long latent period excludes the group of cases, not uncommon, in which secondary paralysis follows redundant callus formation, vicious union, or other mechanical complications of fracture which jeopardize the integrity of neighboring nerve structures.

The curious, and at the same time characteristic, feature of the late paralysis is its appearance so long after reception of the original injury that a direct connection between the two conditions might be open to question, and a natural skepticism as to the etiologic relationship may lead to diagnostic doubts and errors.

He reports three cases:

Case 1: Fracture of the elbow-joint at the age of 5 followed by cubitus valgus and deformity in region of the internal condyle. Thirty-six years later without apparent exciting cause, there was slow development of ulnar neuritis, with atrophy, pain, paresthesias and disturbance of epicritic sensibility limited to the distribution of the ulnar nerve.

Case 2: A saleswoman, aged 65, fractured right elbow at the age of 16; fair recovery with insufficient power of flexion. At the age of 55, that is, thirty-nine years after the injury, symptoms of ulnar neuritis appeared and progressed gradually, namely, weakness, atrophy and disturbance of epicritic sensibility limited to the

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(6) Jour. Amer. Med. Ass'n., Jan. 1, 1916.

ulnar distribution of the hand. There is cubitus valgus, the ulnar groove is shallow and deformed and the nerve is thickened.

Case 3: Fracture of the elbow-joint at the age of 16. Six years later there was gradual development of ulnar neuritis with atrophic paralysis and epicritic anesthesia. Palpation shows cystlike tumor in the ulnar groove associated with enlargement and deformity of the internal condyle. Operation, with removal of a cyst. Pathologic diagnosis—chronic bursitis.

He concludes:

The tardy or late paralysis of the ulnar nerve is a well-defined clinical type of chronic interstitial neuritis, consequent on old injuries and deformities about the elbow-joint.

The interval of time elapsing between the reception of the original joint lesion and the onset of neuritic symptoms may vary from six to thirty-nine years.

During this latent period there are no symptoms other than those referable to the deformity and restricted movements of the elbow-joint.

The joint lesions most commonly associated with this form of neuritis are those resulting from old fractures and dislocations about the elbow-joint and very rarely old arthritis.

Especially frequent is the fracture of the external condyle with cubitus valgus and alterations in the configuration of the ulnar groove.

The neuritic symptoms are slowly progressive and run an extremely chronic course. They consist of atrophic paralysis limited to the ulnar distribution with corresponding sensory disturbances. The sensory symptoms, both subjective and objective, may be insignificant and in the earlier stages even absent.

The diagnosis rests on the presence of an old joint lesion and the neuritic character of the symptoms. In differential diagnosis the progressive spinal atrophies and the *hypothenar type of neural atrophy* (compression neuritis of the deep palmar branch of the ulnar) will demand consideration.

Surgical measures are usually necessary to check the progress of the atrophy and restore function. Among

the procedures advocated in the different types of cases are: enlargement and remodeling of the ulnar groove; resection of the thickened portion of the nerve trunk; transposition of the ulnar nerve to the anterior surface of the internal condyle, and supracondyloid cuneiform osteotomy of the humerus to correct the valgus deformity.

**Alcohol Injections of Nerve Trunks in Certain Traumatic Affections.** J. A. Sicard<sup>8</sup> finds alcohol injections useful in: (1) paroxysmal painful affections due to neuritis following wounds when there is no tendency to spontaneous improvement; (2) in certain persistent motor disturbances, notably contractures in the upper extremities.

In the first group particular mention is made of the very frequent post-traumatic painful affections of the sciatic and median nerves which fail to be relieved by filling the nerve and even by section with immediate suture. Injections of 2 c.c. of 60 per cent. alcohol a few centimeters above the lesion has given excellent relief. Curiously enough, in the median nerve cases, while anesthesia became complete there was not only no increase in paralysis, but the patients could use their fingers better than before the injection. In the second group there were eleven cases of various types of contracture of the muscles supplied by the median and musculo-cutaneous nerves.

**The Gluteal Fold in Sciatic Neuritis.** Hildred Carlill<sup>9</sup> in calling attention to the diagnostic sign says that it is well-recognized that the gluteal fold is not formed by the lower and medial border of the gluteus maximus; indeed, this side of the muscular parallelogram crosses very obliquely the fold of the nates as its fibers pass from above, downwards and laterally.

The fold depends on the tension of the fascia and its adherence to the subcutaneous fat, the skin being creased when the thigh is extended upon the trunk—namely, in the erect position of the body. When the thigh is flexed, either in health or in disease, the fold is lost. Disappearance of the fold, in the absence of any alteration in position of the lower extremities, alters the expression of

(8) Rev. Neurol., August-September, 1915.

(9) Lancet, Aug. 19, 1916.

the buttocks. The chief action of the *gluteus maximus* is to extend the hip-joint, more especially in movements which require considerable muscular effort. When the thigh is kept flexed, as in cases of hip disease, for example, the muscle atrophies from disuse.

The gluteal fold varies in degree in normal persons, but as a rule it is well-marked. It is often duplicated and may be seen in triplicate. When it is poorly developed the subject can increase it usually by tightening up his gluteal muscles or by throwing back the trunk. Bending forward or flexing the thigh causes the fold to be obliterated. The influence of flexion or extension is found only in association with tension of the fascia. The fold is well-marked in people who have an excess of fat in the gluteal region, normal or pathologic.

He points out the fact that the gluteal fold may be poorly developed in sciatic neuritis. Commonly it is absent altogether and this may be the case whether the affection is unilateral, bilateral or part of a multiple peripheral neuritis.

**The Achilles Tendon Reflex in Sciatica.** This has been studied by A. Accornero.<sup>1</sup> He first takes up the question of the constancy of this reflex in health. Older writers, Eulenburg, and Berger, believed it was rather inconstant, but since Babinski introduced the method of testing for it with the patient in the kneeling position, it has become generally admitted that it is practically never absent in normal persons. Oppenheim has endorsed this stand and Charpienter found this reflex present in every one of 1,200 soldiers. When the patient is unable to get up on his knees he should be placed on the side with the knee slightly flexed and the foot tested supported by the left hand of the examiner. Accornero, examined 126 patients with sciatica. He found the reflex equal and normal on both sides in sixty-one, diminished on the affected side in thirty-nine, absent on this side in twenty, and increased in three. In three cases of unilateral sciatica it was absent on both sides although no central lesion was demonstrable. In fourteen cases in which the reflex at first was absent or diminished on the

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(1) Rev. Neurol., October, 1915.

attacked side it became exaggerated after the pain had subsided. There was no relation between the degree of alteration in the reflex and the severity of the case.

### MISCELLANEOUS DISEASES OF THE NERVOUS SYSTEM.

**Nervous Affection of the Sixth and Seventh Decades of Life.** I. Mitchell Clarke<sup>1</sup> discusses the nervous diseases of this age under their various subdivisions.

Of the spinal cord it may be said that the diseases of the spinal cord between 50 and 70 years of age are both numerous and important. Tumors of the cord present no special features at this age; secondary metastases in the cord itself to cancer elsewhere are very rare.

Myelitis is not a common disease after the age of 50. In 156 cases reported as myelitis, acute or subacute, excluding pressure myelitis, 18 per cent. occurred after this age. This relative infrequency after 50 might be expected, as the common causes of myelitis are chiefly some acute infection or intoxication, in which the myelitis may be either primary or secondary. The term "myelomalacia" is more accurate than "myelitis" for many cases. One might perhaps have anticipated an influx of cases of myelitis, or rather myelomalacia, in later life, due to vascular occlusion from arterial degeneration, but large patches of softening in the cord seemed rarely to result from this cause.

Arteriosclerosis in the cord seems to affect the smaller vessels and to produce small sclerotic foci or diffuse areas of degeneration. The diseases of the spinal cord which are especially characteristic of this age are diffuse lesions of the posterior or lateral columns, mostly degenerative—cases formerly grouped together as the "combined sclerosis." Such are the degenerations of the posterior columns found in cachectic states, in carcinoma, in glycosuria or diabetes, the slighter forms of the cord degenerations of pernicious anemia, and also those found in some cases of chronic cystitis and enlarged prostate. In all, the lower part of the cord is solely affected, and

(1) *Lancet*, Nov. 6, 1915.

the predominant symptom is paraplegia or paraplegic weakness with ataxia, with or without some, generally not profound, sensory affection.

He found that in 114 cases of spastic and ataxic paraplegia, combined with sclerosis and subacute combined degeneration, 38 per cent. occurred between 50 and 70 years of age. Hematomyelia of spontaneous and non-traumatic origin is rare at any age. Weakness in the legs or paraparesis is occasionally a symptom of chronic uremia in elderly persons.

Senile paraplegia is a name used for paraplegic weakness due to diverse pathologic changes. For the most part the sufferers are beyond the age under consideration. A variety has, however, been described by Oppenheim which he attributes to arteriosclerosis of the cord, and to perivascular sclerotic changes, especially in the white matter. The symptoms are those of a slowly progressing spastic paraparesis, ultimately leading to contraction of the limbs with increase of deep reflexes. Spastic paraparesis or even paraplegia may in later life be due to cerebral affections, for instance, small patches of softening on each side of the pons, or bilateral lesions of the cortical leg centers, causing degeneration of the pyramidal tracts.

*Pressure Paraplegia:* Caries of the spine, hypertrophic cervical pachymeningitis and internal pachymeningitis involving a great extent of the cord, and probably of syphilitic origin, are very seldom seen after fifty. The most important cause of compression paraplegia in later life is cancer of the vertebrae; the patients are almost all over fifty, and the growth is nearly always secondary. The primary tumor is most frequently in the breast, but may be in the prostate, stomach, uterus or other organ. A rare cause of pressure paraplegia in later life is a limited osteo-arthritis of the vertebrae. Various forms of osteo-arthritis of the spine are well known, and the author refers only to those cases in which a paraplegia, generally painful, is caused by an osteo-arthritic lesion limited to a few vertebrae.

*Tabes Dorsalis:* Tabes dorsalis, the most common disease of the spinal cord, fairly maintains its frequency in living over the age of 50 years was 24 per cent. Byron

living over the age of 50 years was 24 per cent. Byron Bramwell's statistics give the age of onset in 263 cases; 9.6 per cent. occurred in patients over 50 and 1.9 per cent. in those over 60 years. Cases in which the onset occurs after 45 as a rule run a more favorable course. Tabes shows a decided falling off after 50 years as compared with the age period 30-50, and a very marked fall after 60. The cases over 50 comprise: (1) stationary and latent cases; (2) others only slowly progressive; of these the onset in 30 per cent. took place between 40 and 50 years; (3) cases which begin after 50. In some of these there was a late syphilitic infection after 40 years of age, but in others the interval after infection was from twenty to thirty years or even longer.

The myopathic forms of muscular atrophy do not affect the period of life under consideration. Of 100 patients with various forms of muscular dystrophy three or 3.5 per cent. were over 50 years of age. On the other hand, the myelopathic forms, progressive muscular atrophy, bulbar paralysis, and amyotrophic lateral sclerosis, are relatively frequent after 50. The order of frequency of these diseases after 50 is: (1) bulbar paralysis, in which the large majority of cases occur after this age; (2) progressive muscular atrophy; and (3) amyotrophic lateral sclerosis. In sixty cases of progressive muscular atrophy 30 per cent. occurred between 50 and 70, and of sixty-three cases of amyotrophic lateral sclerosis 12 per cent. were found between these ages. Some rare cases of progressive muscular atrophy run a subacute course, and are especially met with in the elderly. Lastly, may be mentioned the very occasional occurrence of atrophy of the small muscles of the hand in connection with a cervical rib even so late as 50 years of age.

Disseminated sclerosis is essentially not a disease of the period of life under consideration. It generally begins much earlier, but as it may run a long course there will be a certain number of survivors after 50.

In 536 cases from various sources irrespective of age of onset the number over 50 years was from 4 to 5 per cent.

In syringomyelia the majority of cases occur between 20 and 45 years of age. The disease, however, is encoun-

tered at all ages, and considering that it is one of the rarer nervous diseases, fairly often over 50. Among 135 patients 23 per cent. were between 50 and 70.

In 250 cases of multiple neuritis from various sources only 10 per cent. occurred over the age of 50, and 75 per cent. of the patients were between the ages of 30 to 50.

In forty-five cases under the author's care twenty-four, of whom eighteen were women, were between the ages of 40 and 53 years. There seems to be a special incidence of alcoholic neuritis during the climacteric.

Of the less common forms of multiple neuritis, glycosuria is perhaps relatively the most frequent after 50. The etiology is often mixed—gout, glycosuria, and alcohol.

It remains to mention the cachetic forms of neuritis, and especially that which develops in connection with carcinoma, for the majority of such cases fall between the ages of 50 and 70. The symptoms as a rule are of slight intensity. Toward the end of this period the senile form of multiple neuritis, which in some instances is referable to arteriosclerosis or atheroma, appears.

Meningitis due to pus-producing organisms becomes uncommon after 50; when it occurs as a primary infection the diagnosis is now greatly facilitated by lumbar puncture. Perhaps the most frequent form is the pneumococcus, which may either be primary, or a complication of pneumonia, or one feature of a general pneumococcus infection.

Tuberculous meningitis in persons over 50 is very exceptional. Of 560 patients with tuberculous meningitis, four had passed this age.

The most common of all nervous affections between 50 and 70 years of age are those due to disease of the cerebral vessels, and the causes and methods of prevention therefore belong to diseases of the vascular rather than of the nervous system. Of 500 cases of cerebral hemorrhage, the onset was between the ages of 50 and 70 in 321, or 64 per cent., and of 110 cases of cerebral thrombosis, in sixty-seven, or 60.9 per cent.

An intracranial tumor is rarely met with in persons over 50, and still more rarely in those over 60; when it

occurs diagnosis may be more difficult than in younger persons.

Of 261 individuals with cerebral syphilis, 9 per cent. were over 50 and only three were over 60 years of age; these include sixty personal cases, of which the number over 50 was eight.

Syphilitic affections of the spinal cord are relatively still more uncommon after fifty years than those of the brain.

Speaking generally, migraine is uncommon, at any rate in a severe form, after 50.

It is well known that the frequency of epilepsy diminishes as life goes on, but it is also true that epilepsy may begin at any age. Of 100 consecutive out-patient cases 6 per cent. had their first fit after 50, and in 100 consecutive private patients 9 per cent.

In late epilepsy men are more frequently affected than women. Idiopathic epilepsy between the ages of 50 and 70 comprises: (1) cases in which the disease persists throughout life; (2) cases in which the patient suffered from fits in childhood or early life, and then was free for a number of years, with subsequent reappearance late in life; and (3) those in whom the onset is after 50.

The first occurrence of epileptiform fits after 50 years of age may be connected with the presence of arteriosclerosis and a high blood-pressure. These cases can not therefore be brought into the category of idiopathic epilepsy.

Vertigo is one of the common and certainly one of the most distressing, nervous affections of later life. By far the most frequent form is aural vertigo. Next in frequency come the cases due to increased intracranial pressure. It is also met with over 50 as a symptom of atheroma of the cerebral vessels, in stenosis of the aortic valves, in granular kidney, in cerebral tumors, and in neurasthenia.

The more severe hysterical affections are undoubtedly uncommon after 50.

The majority of cases of neurasthenia occur between the ages of 25 and 50. Many neurasthenics, as they reach later life, though perhaps never becoming robust, yet arrive at a measure of health and freedom from

morbid anxieties. At the same time neurasthenia, thought not frequent, may occur after 50 up to old age.

Paralysis agitans, though not a very common disease, is pre-eminently one of the period of life between 50 and 70 years. Its main incidence is narrower and lies between 50 and 65.

The traumatic neuroses are apt to affect persons after 50 very severely as the result of an accident, and to take the form of neurasthenia rather than of some more strictly hysterical manifestation.

Between 50 and 70 true neuralgia is not an infrequent affliction. Gout, arteriosclerosis, various cachexias, and presenile disturbances of nutrition are the chief causes. The senile and arteriosclerotic forms chiefly affect the fifth nerve.

Among his concluding remarks Clarke says: "Lastly, the purely nervous diseases which are especially characteristic of this period of life are paralysis agitans, some of the combined scleroses of the spinal cord, and certain muscular atrophies of central origin. It is possible that some of these affections owe their origin to that process of premature senescence or defective vital endurance of certain neuronic systems known as abiotrophy, and that this pathologic process may occur at any age. Paralysis agitans especially, in its narrow age incidence, uniformity of symptoms, steadily progressive character, and occasional familial tendency, suggests this mode of origin."

**Progressive Lipodystrophy.** Charles Herman<sup>2</sup> reports a case of progressive disappearance of adipose tissue of the face, upper chest and arms. This condition began at the age of 6, and at 11 the fat of the face had almost entirely disappeared. Herman reviews the literature collecting fifteen cases; all of the undated ones being in females. The condition occurs both in married and unmarried women who present no unusual disturbances of menstruation, pregnancy or lactation (see Plate X.).

The loss of the subcutaneous fat of the face begins insidiously without fever, pain or other symptoms, and gradually becomes more marked; it is bilateral and symmetrical. In six of the patients, including his own, the disease began between the fifth and the seventh year; in

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(2) *Archiv. Int. Med.*, April, 1916.

the remainder, about the time of puberty. In the majority of the cases the loss of subcutaneous fat then extended to the neck, arms and upper part of the trunk. The orbital fat is little if at all affected and as no post-mortem examinations have been made it is still unknown whether the internal fat is diminished. The increase of adipose tissue in the lower part of the body begins or becomes more marked at the time of puberty. This increase is most distinct in the gluteal region and on the outer side of the thigh, but also involves the rest of the leg. As a rule, both sides are equally affected; occasionally the increase is greater on one side.

Ernst Jolowicz<sup>3</sup> reports a case of an unmarried woman whose face, arms and chest aggressively and slowly became lean from the age of 8. At the same time an accumulation of fat occurred about the hips and thighs. He says that the term lipodystrophia progressive suggested by Simons does not entirely conform with the course of the disease, inasmuch as in his case the condition came to standstill in childhood. He is of the opinion that it is an isolated trophic change in the subcutaneous fat and agrees with Simons<sup>4</sup> that there is a possible disturbance of the endocrinous glands.

Gerstmann<sup>5</sup> describes a case of progressive lipodystrophy. There are only sixteen cases on record to his knowledge. His patient is a man of 32 and the lipodystrophy was first noticed at the age of 10. This, he says, seems to be the first case known in a man. The face, arms and trunk are exclusively affected; the thyroid is enlarged, and there are symptoms indicating excessive thyroid functioning.

**Experimental Studies of Injection of the Gasserian Ganglion Controlled by Fluoroscopy.** Lewis J. Pollock and Hollis E. Potter<sup>6</sup> state that from a study of forty skulls they found that the alveolar foramina were less than the alveolar tubercular measurement by from 0.1 to 0.9 cm. Eighty per cent. of the cases showed a difference

(3) Neurol. Centralbl., Dec. 16, 1915.

(4) Segmentary adiposis of the lower extremities showing a similar appearance to Simon's case, but in which no mention is made of lipodystrophy is shown in the Practical Medicine Series, Vol. X, 1913, p. 178.

(5) Wien. klin. Wochenschr., Sept. 21, 1915.

(6) Jour. Amer. Med. Ass'n., Nov. 4, 1916.

of less than 0.6 cm., 50 per cent. less than 0.4 cm. Of the cases showing a difference of over 0.6, many occurred where the intertubercular diameter was less than 5 cm., and some where the alveolar tubercular measurement exceeded 5.5 cm. The depth of the foramen from the zygoma did not bear a constant relation to the alveolar foraminal measurement, except in that where the foramen was deeply situated the alveolar foraminal measurement was often inclined to be greater than usual.

They found that the anterior border of the petrous portion of the temporal bone, when the head is viewed exactly laterally through the fluoroscope, throws a shadow easily seen in living subjects. This shadow forms a line which is a direct projection of the shadow cast by a needle entering a point between the second and third molars of the upper jaw. If, therefore, a needle be introduced here and pushed, according to Härtel's directions, to a depth computed by their measurements, it will be approximately at the foramen ovale. If now the needle be directed so that a projection of the shadow would follow the shadow of the anterior border of the petrous portion of the temporal bone, or 1 or 2 mm. below it, the anteroposterior and superior planes of the foramen ovale will be accurately located. The internal and external planes of the foramen ovale cannot be located by the fluoroscope, and one is guided by Härtel's direction to point at the pupil of the same eye and by the pterygoid bone. The superior inferior plane may be further determined, if necessary, by employing a shadow of an opaque eustachian bougie, when the needle should be above it, and here the secondary rays do not interfere with the visibility of the shadow of the needle.

In conclusion they say that employing the shadow cast by the anterior border of the petrous portion of the temporal bone, on a fluoroscope, as a line of orientation in injection of the Gasserian ganglion by Härtel's method is made more certain (see Plate XI.).

**Metabolism in a Case of Myasthenia Gravis with Consideration of Administration of Calcium and of Glandular Preparation.** Arthur Bookman and Albert A. Epstein' report on studies in metabolism in a case of

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(7) Amer. Jour. Med. Sci., February, 1916.

myasthenia gravis presenting as unusual symptoms bronzing of the skin and pigmentation of the buccal mucous membrane and high blood-pressure.

Except for the creatinin, there was no striking variation from the normal in any of the constituents studied during any of the experiments. There was throughout a retention of calcium of magnesium, and of sulphur, and except for a very slight loss in the last period, a substantial retention of nitrogen and phosphorus. There was no disturbance of the ammonia excretion even during the exercise period (Period 2), although at this time there was less retention of nitrogen. There is, therefore, contrary to the opinion held by Kaufmann, no evidence of an acidosis in this case. During thymus feeding there was an increased retention of nitrogen and of phosphorus. Three days before the beginning of the period of calcium administration the patient had a slight intestinal disturbance for two days, accompanied by abdominal cramps and diarrhea. It is probable that the slight loss of nitrogen and phosphorus is connected with this. During this period almost all of the additional calcium fed to the patient was excreted.

In common with previous observers the authors found a low creatinin excretion. This shows best in the creatinin co-efficient, which averages 4.8 mgs. creatinin-nitrogen per kilo (normal 7-11 mgs.), while the ratio excreted to the total nitrogen averages 3.6 per cent., (normal 3.9 per cent.). During the thymus period and the calcium period creatin was excreted in noticeable amounts. In two cases of myasthenia, Halpern found marked retention of nitrogen, phosphorus and lime, with a normal ammonia excretion. Spriggs had found the creatinin excretion decidedly below normal in his patient. Pemberton was able to corroborate this in one case, in which he found also a marked loss of calcium with a normal nitrogen balance. Diller and Rosenbloom have recently published a case in which they got the same results for calcium and creatinin, accompanied, however, by a negative nitrogen balance. Kaufmann in his patient investigated principally the nitrogen metabolism and the ammonia excretion. He found that exercise caused a decrease in the retention of the nitrogen and if continued,

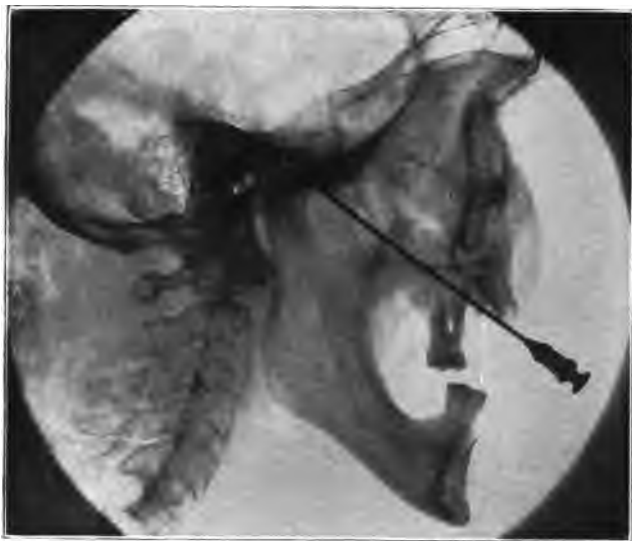
PLATE X.



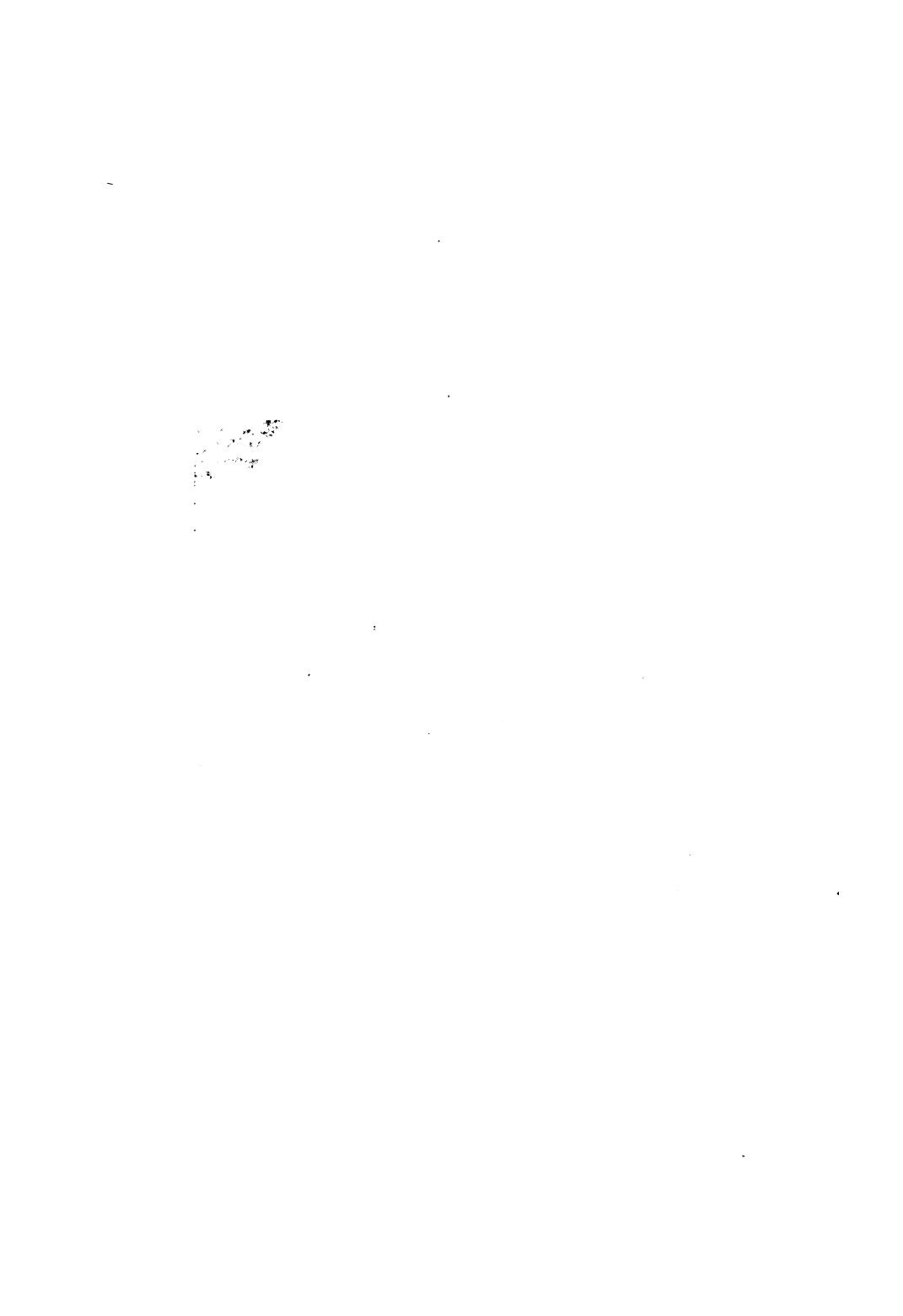
Progressive lipodystrophy in a woman of 21 years. Simons' case.—Herman  
(see page 165).



PLATE XI.



Needle inserted through the foramen, resting on the anterior border of the petrous portion of the temporal bone.—Pollock and Potter (see page 166).



a loss together with increased excretion of ammonia. In his case there was, however, disease of the liver.

Ovarian and testicular substance were also administered to their case. Neither the glandular preparations nor the calcium appeared to have any influence upon the clinical course.

**Periostitis and Osteitis as a Cause of Local Pains and So-Called Neuritis and Neuralgia.** Charles L. Dana<sup>s</sup> says:

"If by neuralgia is meant a chronic or subacute malady, functional in nature and characterized by pain along a nerve, one must limit its existence to a few types and call it a rare disease.

"In recent years some neuralgias, notably sciatica, have been shown to be of arthritic origin and character; and the routine treatment of sciatica now is to bind and immobilize the limb and pelvis. A genuine sciatic neuritis is a rather rare disease. And we have mostly nowadays to deal not with varieties of typical neuralgia but with local pains of occipital, brachial, intercostal, lumbar, crural, or sciatic distribution.

"It has been found that some of these are due to infections attacking joints, muscles and tendons. In certain cases these persistent pains are due to osteoperiosteitis of acute infectious origin."

The conclusions which he draws from these experiences are:

1. Acute infections from the teeth, tonsils, and other foci may cause local forms of osteitis and periosteitis.

2. This fact must be reckoned with in diagnosing neuralgia and other persistent and distressing local pains.

3. The joints are not the only regions attacked by acute infection; and the arthritis explanation of pains in the back and extremities has been perhaps overdone. So-called "sciatica," for example, is not always arthritic or myositic or neuritic.

4. Further study is needed to corroborate these findings of Dana's, and to determine the source and nature of the infections that produce them.

5. Syphilis and tuberculosis do not play so large a

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(8) Med. Record, April 29, 1916.

part as supposed in the causation of these minor types of osteoperiosteitis.

6. "Neuralgia" as usually defined is a very rare disorder, and its diagnosis is to be made only after careful examination of muscles, bones, nerves, nerve centers, and sources of reflex irritations.

**Paralysis During Pasteur Antirabic Treatment.** Frank S. Fielder<sup>9</sup> reports seven personal cases, one terminating fatally, and six other previously unreported cases.

He says this complication occurs with great rarity, and usually terminates in recovery, but the possibility of paralysis should always be explained to those in whom infection is doubtful, or who are inclined to insist on being treated merely as a sentimental matter of precaution.

The symptoms vary from a slight degree of neuritis, with little or no motor involvement, to an acute ascending paralysis which may be fatal.

The frequency of paralysis as a complication of antirabic treatment is variously estimated by different writers, but its real frequency is unknown, since many cases, especially the mild ones, are doubtless not reported.

His seven personal cases, together with six other cases which are here first reported, bring the total up to 143 cases with twenty-five deaths.

In his thirteen cases the onset averaged twenty-six days after the bite and seventeen days from the beginning of treatment.

The onset of the symptoms is usually acute, with pain and numbness of the affected parts, followed by muscular weakness. Prodromal symptoms if present are mild, and consist only of malaise and nervous depression, with perhaps some restlessness, insomnia and anorexia.

Males seem to be affected much more frequently than females. Young children are rarely affected. Most of the cases occur in adults of varying ages.

He concludes:

1. Paralysis complicating the Pasteur antirabic treatment is very rare, but doubtless occurs in mild form more

(9) Jour. Amer. Med. Ass'n., July 3, 1916.

often than is known. It affects adults chiefly, young children almost never.

2. The prognosis is good in cases of mild or moderate degree, but the mortality among the severe cases is high.

3. The administration of antirabic treatment is therefore not entirely devoid of risk.

4. The possibility of the occurrence of paralysis in any particular case, however, is so slight that it should not deter those who evidently need antirabic treatment. A person bitten by a rabid animal is much more likely to develop rabies, which is certainly fatal, than paralysis, which usually ends in recovery.

5. The slight risk of paralysis should always be explained to those who are inclined to insist on antirabic treatment in the absence of clinical or laboratory evidence that the biting animal was rabid.

6. In most instances this paralysis is caused by the antirabic treatment itself (fixed virus infection, or toxin, or both). It is not often due to street virus infection modified by the treatment. Its incubation is shorter than that of most cases of street virus rabies.

7. Variations in the strength of the fixed virus used are probably a factor in the etiology.

8. Personal idiosyncrasy is an important predisposing condition.

**Results of Neurologic Studies of the War.** H. Oppenheim<sup>10</sup> presents some of the unusual phases of war injuries. His experience regarding late abscess differs from Marburg's, as to the frequency. In commenting on gunshot wounds of the brain he says that the insignificance of the focal symptoms as compared to the extent of the bullet wound is notable. On the other hand, the multiplicity of focal symptoms in some cases speaks for numerous lesions in the central and peripheral tracts which were struck by the bullet in its course. Cortical symptoms prevail in cases of tangential, grazing and superficial wounds.

He calls attention to the predominance of bilateral manifestations, of which cerebral paraplegia is a special syndrome. In the motor disturbances the phenomena

(10) Berlin. klin. Wochenschr., November, 1915.

followed definite rules, whereas sensory disturbances frequently did not conform to such rules. It was remarkable to find the great number of unilateral and bilateral disturbances of vision. The frequency of cerebral and labyrinthine symptoms as the result of remote gunshot wounds are probably explained on the basis of fissure formation.

He calls attention to Weber's description of a characteristic disturbance of the consensual vascular reaction in lesions of the cortex. This manifests itself by the fact that temperature stimulation affected not only the textured areas but produced an increased vessel reaction in the whole skin area of the body.

In gunshot wounds of the spinal cord the most common result was circumscribed neurosis, which formerly was diagnosed as hemorrhage. Brown-Séquard paralysis with homolateral anesthesia was not very rare. It is caused by the fact that the penetrating bullet on one side grooved the motor and on the other the sensory tract. While the typical spinal cord lesion is partial or complete transverse section in some instances a longitudinal destruction has been found. He points out the occurrence of combination of cord and plexus injuries where operative treatment offers doubtful results. In some cases the injury to the spinal cord was so slight that in one case the only symptom found was Babinski's sign on one side; in another paresthesia in the distribution of the ulnar nerve, and in another disappearance of the deep reflexes of one side.

In cases of injuries of the ischiatic nerve the sensory fiber (plantar pedis), or motor fiber alone may be injured. In injuries affecting the median distribution, the flexor indicis, the radial, the extensor digiti communis, and the upper brachial plexus, the deltoids were involved to the greatest extent. The ulnar nerve has greater influence upon the thenar eminence than was formerly recognized and the pronator radii teres is not always innervated by the median nerve.

As to electrical reaction he has found many cases in which sluggish reaction to the galvanic current occurred later than was expected. In certain lesions of the peripheral nerves electrical stimulation of the distal por-

tion caused no contraction, whereas stimulation of the peripheral end produced movement showing that conductivity may be preserved when stimulability is lost. It has been shown that the first sign of return of function after nerve suture is the consensual vessel reaction to temperature stimulation. Many patients have shown some hyperthyroidism, as Graefe's sign, tachycardia, hyperidrosis and tremor as signs of traumatic neurosis.

Attention has been called to the predominance of left-sided symptoms; perhaps according to Leipman because of the lesser resistance of the right hemisphere to physical and psychical shock.

Akinesia amnestica, and reflex paralysis are terms offered by Oppenheim to the condition called local traumatic hysteria.

These types of paralysis show in their manner, distribution and in the way the injured limb is moved certain definite rules. It is especially notable that the paralysis of the leg nearly always increases distally and in the distal muscles, especially the peroneus, the paralysis remains the longest.

**The Effect of High Explosives on the Central Nervous System.** Fred W. Mott<sup>1</sup> in the Lettsonian Lectures deals exhaustively with the above subject. He states that the effects of high explosives on the central nervous system fall into three groups.

1. In Group 1 are the immediately fatal cases, either from pieces of shell, stones, rocks, or portions of buildings striking the individual, causing instant death, or the person may be buried from the explosion of a mine. Again, instant death must have occurred in groups of men from the effects of shell fire and yet no visible injury has been found to account for it.

2. In Group 2 we can place those cases in which the detonation of high explosives has caused wounds and injuries of the body, including the central nervous system, which have not been immediately fatal. The number of these cases which do not exhibit any of the functional disorders and disturbances characteristic of what is termed "shell shock" without visible injury, although such individuals have received most serious and fatal

(1) *Lancet*, Feb. 12 and 26, March 11, 1916.

wounds from exploding shells, leads one to consider that in a large proportion of cases of shell shock without visible injury there are other factors at work in the production of the nervous symptoms besides the actual aërial forces generated by the explosive.

3. The third group includes injuries of the central nervous system without visible injury, and to this group Mott has given especial attention, as it is the one of which he has had most experience. The causes of shock to the nervous system by high explosives may be considered under the headings of physical trauma—concussion or "*commotio cerebri*" by direct aërial compression or by the force of the aërial compression blowing the person into the air or against the side of the trench or dug-out; or by blowing down the parapet or roof on to him causing concussion; or a sandbag hitting him on the head or spine might easily cause concussion without producing any visible injury. Again, he might be buried and partly asphyxiated or suffer from deoxygenation of his blood by carbon monoxide poisoning, for these high explosives generate considerable quantities of CO which is inodorous and would not be recognized.

If aërial concussion by the forces generated by high explosives can cause death without visible injury, Mott thinks that more probably it would arise from sudden arrest of the medullary centers. The stem of the brain surrounded by the cerebrospinal fluid is prevented from oscillating by the nerves which issue from it to pass through the holes in the skull; likewise the spinal cord by the anterior and posterior roots and the ligamentum dentatum is prevented from oscillating. A sudden shock of great intensity would be transmitted through this incompressible fluid, and seeing that it not merely surrounds the central nervous system but fills up the ventricles and central canal and all the interstices of the tissues serving as it does the function of lymph, it follows that a shock communicated to the fluid of sufficient intensity would make itself felt on all the neurons.

Severe concussion can not only cause immediate dissociation of the cortical preceptor neurons, producing unconsciousness or a disturbance of conscience, but for a varying period of time it can destroy the power of

recollection of perceptions prior to the shock. There is a retrograde amnesia, and in very severe cases of shell shock, there may be a complete loss of memory both as regards recollection and recognition.

The delicate granules filling the nerve cells have been termed "neurobions," as if they were independent units, but this is theory. It is, however, conceivable that violent concussion transmitted to the cerebrospinal fluid, which forms the circumambient medium of such a complex mechanism as the living nerve cell, would cause a violent oscillation of these neurobions and a loss or disturbance of their functions or variable duration according to the severity of the shock.

It is known that a continuous supply of oxygen is essential for consciousness. Now a violent emotion such as fright can by its influence on the vasomotor center and the heart's action, causing a fall in the blood-pressure, produce an immediate lowering of oxygen tension in the fluid, and thereby suspension of function of the intercalary neurons of the cortex, followed by dissociation of the cortical perceptors and loss of consciousness. In many of the disorders of functions and loss of functions of the central nervous system resulting from shell-shock, using that term in its widest sense, there occur symptoms of cortical dissociation—*e. g.*, cortical blindness, deafness, mutism and paralysis.

"The symptoms of CO poisoning so accord with those functional disorders of the central nervous system which have so frequently been found to occur in shell shock with burial that one naturally thinks it possible that while lying unconscious at the bottom of a trench or dug-out sufficient CO is inspired to cause the severe effects on the mind which some of these patients exhibit. In some cases there are marked tremors and when intentional and accompanied by other signs a condition of disseminated sclerosis may be considered."

Commenting on the probable pathogenesis he says: "M. Arnoux's theory is, then, that the sudden increase of atmospheric pressure produced by the explosion is capable of producing an immediate increase and absorption of air and CO followed by a sudden liberation on return to normal conditions. Suppose the air is charged

with carbon monoxide and oxides of nitrogen, would it not be possible for the man to inspire enough of these gases to cause instant death?" Mott wrote to Professor Leonard Hill on this subject, and received the following very interesting reply:

"The explosion of a big shell in a trench, dug-out, cellar, or other confined space must, I think, instantly deoxygenate the air and produce a high concentration of carbon monoxide and oxides of nitrogen. The inspiration of a man at the moment of explosion may introduce enough of these gases to cause death from want of oxygen. If he is fatigued his muscles will be in the condition to go into rigor on the sudden deprivation of oxygen."

Mott recapitulates the possible effects of the detonation of high explosives on the nervous system in cases where there is no visible external injury. They are:

"1. Commotion from the aërial compression. 2. Concussion with or without burial. 3. Decompression with embolism, by bubbles of nitrogen and carbon monoxide. 4. Inspiration of carbon monoxide during the aërial compression. 5. Prolonged inhalation of noxious gases—*e. g.*, carbon monoxide—while lying unconscious or partially buried.

"The mental and bodily condition of the individual at the time of the shock may be classified as follows: (1) inborn: (*a*) a timorous disposition and an anxious temperament, (*b*) a neuropathic or psychopathic inheritance; (2) acquired: (*a*) a *locus minoris resistentiae* in the central nervous system in consequence of alcoholism, syphilis, or previous head injury, (*b*) a lowered neuro-potential, the result of a post-febrile neurasthenia, (*c*) nervous exhaustion the result of mental stress, anxiety, insomnia, and terrifying dreams, (*d*) bodily exhaustion from fatigue, cold, wet and hunger.

"It will be observed that a large majority of the cases of so-called shell shock admitted with functional neurosis in some form or other occurred in individuals who either had a nervous temperament or were the subjects of an acquired or inherited neuropathy.

"It will be observed that out of 156 cases in which a previous history is reported in the notes, fifty-two gave a history of either a previous nervous breakdown or a

timid disposition, easily frightened, emotional, or afraid of the sight of blood; in a few the fact was elicited that they had had a fright in early childhood and that this recurred in dreams. Some gave a history of neuropathic tendency or inheritance.

"The symptoms of shell shock among them are as follows:

"Loss of memory may be a complete loss of power of recollection and of recognition. Consciousness, except for the immediate present perception, may be a blank, and there is in such cases, as a rule, no art to find the mind's complexion in the face, for a patient so afflicted exhibits a dazed, mindless expression.

"The frequency with which these men with shell shock suffer with terrifying dreams at night and in the half-waking state points to the conclusion that a psychic trauma is exercising a powerful influence on the mind by the thoughts reverting to the terrifying experiences they have gone through, and their continuous influence on the subconscious mind may account partially for the terrified or vacant look of depression on the face, the cold blue hands, feeble pulse and respiration, sweats and tremors, some or all of which signs of fear the severer cases manifest.

"Various forms of speech defects are common; they are mutism, aphonia, stammering, stuttering, and verbal repetition. The most frequent speech defect is mutism. About one in twenty of men admitted with a history of shock due to high explosives, and having no visible signs of injury, suffer with mutism, but nevertheless are quite able, as a general rule, to write a lucid account of their experiences. Most of the men so afflicted are unable to whisper or produce any audible sound; thus there is no sound when they laugh. They are unable to whistle or to cough, and in severe cases there is difficulty of putting out the tongue and, in one case, of swallowing."

Mott supposes that mutism is caused by fear producing an emotional shock depressing the activities of the whole of the cortical structures connected with the phonation and production of audible sounds.

Of headaches he states that:

"So long as the patient is in a state of shock he feels

heavy and dazed and does not suffer great pain as a rule, but later, as consciousness becomes less clouded, so the headache which invariably follows shell shock becomes more acute.

"The commonest situation for the maximum pain is the occipital region and the back of the neck; it is often described as a tight compression like a helmet—the helmet of Minerva.

"The patients often complain of palpitation, breathlessness on exertion, and precordial pain. There may be physical signs of dilatation and tachycardia. The pulse is often small and increased in frequency: the blood-pressure is never high.

"Cutaneous and deep sensibility of the body may be affected, and in severe cases of shock I have occasionally found loss of skin sensibility to all forms of stimulus, pricking, heat, cold and touch, also of deep sensibility, pressure of muscles, movement of joints, and bone sensibility to vibration of tuning-fork.

"Hearing, like speech, and often with speech, is completely lost, so that there is a condition of functional deaf-mutism. Sometimes speech returns before hearing, or the converse may happen.

"Hyperacusis, or extreme sensibility to sound, is a common and very troublesome symptom, making the patient miserable and apprehensive; it also excites or aggravates headache.

"Vision: The sight may be greatly affected during the acute stage, but it is commoner for the patient to complain of 'smoky vision.' Again failure of accommodation and sluggish light reflex are not uncommon in the acute stages.

"Hyperesthesia is a very common symptom.

"Tremors are extremely common and constitute a serious disability; they are coarse and fine, continuous during the waking state, absent in sleep; they may be general, affecting arms, legs and head; they may affect one-half of the body, both lower limbs or both upper limbs.

"Functional paralyses are not at all uncommon, the most common being paraplegia, but hemiplegia and monoplegia are also frequently met with."

Charles S. Myers<sup>2</sup> commenting on certain cases of shell shock treated by hypnosis says:

"Despite the slow progress recorded in certain cases, I have not the least doubt that the hypnotic treatment which these and other cases received here invariably proved of great assistance toward recovery, and would have proved of still greater value if it could have been occasionally repeated later. It may be argued that mutism, rhythmical spasms, anesthesiae, and similar purely 'functional' disturbances disappear after a time without specific treatment. But no one who has witnessed the unfeigned delight with which these patients, on waking from hypnosis, hail their recovery from such disorders can have any hesitation as to the impetus thus given toward a final cure. More especially is this the case in regard to the restoration of lost memories. Enough has been already said about the striking changes in temperament, thought, and behavior which follow on recovery from the amnesia.

"This much may be taken for granted here, that the restoration to the normal self of the memories of scenes at one time dominant, now inhibited, and later tending to find occasional relief in abnormal states of consciousness or in disguised modes of expression, such restoration of past emotional scenes constitutes a first step toward obtaining that volitional control over them which the individual must finally acquire if he is to be healed. Thus the minimal value that can be claimed for hypnosis in the treatment of shock cases consists in the preparation and facilitation of the path toward a complete recovery."

Nervous disorders arising in soldiers from violent shell explosions are, according to Soukhanoff,<sup>3</sup> in the majority of cases due to organic causes. Though no external signs of lesions can be found, no wounds of the skin or head and no fractures, etc., the resulting symptoms such as mental confusion, depression, amnesia, ear and eye disturbances, paralysis, etc., are due to real anatomic changes in the central nervous system. This assumption is sustained by the findings in the spinal fluid such as

(2) *Lancet*, Jan. 8, 1916.

(3) *Russkiy Vrach.*, Oct. 3, 1915.

blood, abnormal albumin content and lymphocytosis, and the subsequent wasting of muscle. Psychogenous and hysteric symptoms may complicate the clinical picture, but they are of secondary nature. Therefore, Soukhanoff thinks that the disturbances should not be entitled "contusion psychoneurosis" but should be designated as air traumatism of the brain and spinal cord. The pathogenesis of these lesions may be explained partly by the action of the rarefied atmosphere from the passing shell and partly by the formation of emboli in the blood, as in caisson disease. The immediate action of the condensed air may hurl the man to the ground and cause a state resembling the so-called concussion of the brain. The poisonous gases resulting from the shell explosion, the emotional shock and, possibly, hemorrhages in the central nervous system from an involuntary violent muscular effort are among other factors liable in this morbid condition.

## PSYCHIATRY.

### GENERAL CONSIDERATIONS.

**The Relation of the Physical and the Psychological to Disease.** E. Bleuler<sup>1</sup> says that only in rare borderline cases is there only one physical or only one psychical cause. The question "physical" or "psychical" should be replaced by: "In what way physical and in what way psychical?" In most of the cases the physical element creates the predisposition; the psychical determines the symptomatology in its details. In one who is predisposed, confinement (prison) leads to psychosis. If there is any form of brain degeneration, certain experiences might produce hysterical symptoms. The epileptic poison makes thinking dull and creates a certain predisposition. Affect and complex determine the specific contents of dream states.

But there are also psychical predispositions, affective experience creates a *locus minoris resistentiae*, so that later psychical traumata might have a pathologic effect.

(1) Zeitschr. f. d. ges. Neurol. u. Psychiat., June, 1916.

Laziness and improper education create nosophilia, an accidental cause makes the disease.

Impeded contact with surroundings (deafness) creates the soil for distrust and sensitiveness, on which through certain experiences, psychosis may develop.

Predisposing and exciting causes frequently work in the same direction and sum themselves up. Or the accident takes advantage of any diseased bodily predisposition to create a disease symptom; a weak stomach is taken advantage of to produce digestive disturbance. The predisposition to vomiting in pregnancy is used to express the distaste for carrying the child of an unloved or ambivalent man. Bodily changes can create the idea of disease which in turn creates bodily symptoms (menstrual disturbances).

A passing bodily disease can create an association which later is hard to dispose of; for instance, pains after fractures and zoster, and tics. The fact that a disease can be influenced in a psychical way does not prove by any means its psychical genesis—fatigue is overcome, pain forgotten, an epileptic attack is suppressed or postponed, etc.

Bodily symptoms do not prove the bodily origin of disease (hysterical diarrhea, menstrual disturbances). It is certain that most of the Schizophrenics, probably all of them are caused by a toxic or anatomic change in the brain, the primary symptom which we know very little about but on which the psychical mechanism brings about most of the symptoms. Manic-depressive insanity is a disease in the main conditioned by physical elements despite the fact it is psychical in nature. In case of organic psychosis, physical processes bring about the largest part of the symptomatology which is modified only to a very small extent by psychical influences. In the neurosis there is nearly always a predisposition on which producing causes liberate the disease, but then only if psychic determinating factors are added, as desire for sickness in case of discontented health, consciousness, fever, habit.

Habits often play the main rôle in children. Occasionally a psychical influence causes a physiologic complex which explains the similarity of many nervous syn-

dromes (globus hystericus, asthenia, diarrhea, vomiting, certain forms of hysterical attacks, epileptic attacks). The exact differentiation of the physical from the psychological causes is of great importance for our therapeutic efforts.

**The Psychiatric Needs of a Large Community.** Owen Copp<sup>2</sup> in an address outlines some splendid plans relative to the perfecting of our systems of caring for the insane in state institutions and in making suitable provision for mental hygiene. He says that close coöperative relations must exist with boards of health, organized charity, reformatory and penal agencies, educational commissions and societies for protection of child life, social uplift and betterment of living and working conditions of the masses, but initiative and independence should be maintained.

"Boards of Insanity should become Boards of Mental Hygiene, expressive of constructive purpose instead of degenerative sequence of disease.

"The field of mental hygiene is so broad and extensive as to suggest the probability that development of its manifold problems and the growth of its activities in their solution may require subdivision into main bureaus of mental *disease* and mental *deficiency*.

"Within the scope of its purposes are:

"A. Acquisition of knowledge of the nature, causes, results, methods of prevention and treatment of mental disease and mental defect.

"B. Interpretation and diffusion of such knowledge for information of the public and the medical profession itself, as to the magnitude and import of the problems of the mentally affected, in order that they may be aroused to consciousness of their obligations and self-interest in promoting, with energy, the aims of mental hygiene.

"C. Prevention of mental abnormality:

"1. By removal of its causes, especially its great and preventable causes, alcohol and drug inebriety, syphilitic infection, and sources of bad heredity through education as to their causative relation, and discriminating application of preventive and remedial measures in accord

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(2) Amer. Jour. Insan., July, 1916.

with enlightened medical and public opinion. Segregation and sterilization of defectives and degenerates, supervision of marriage, etc., would come under consideration in this connection.

"2. By early education in acquiring correct mental habits, in facility of adjustment to difficult situations in life, in fitness of occupation and adaptation to the right levels in social and industrial relations.

"3. By bringing within reach of the indigent, through the family physician and general practitioner of medicine, sufficient knowledge of mental disease for its detection at the earliest manifestation, in order that the best chance of cure may be afforded.

"D. Community care and treatment of the mentally affected:

"1. Registration and supervision of such in the community.

"2. Their temporary care and treatment, pending formal commitment to institutions.

"3. After-care of such patients restored by institutional treatment.

"4. Family care, as an organized system of provision for harmless mental patients.

"E. Institutional provision, whose aims are:

"1. Preliminary observation and treatment, under the voluntary relation, of incipient and borderline mental disease during the period of absence of manifest symptoms of mental disorder and unwillingness of patient and friends to accept hospital treatment, although recognizing illness and need of removal from home environment for rest and recuperation. Such are the functions of the *Preventorium*.

"2. Cure of mental disease, or its amelioration, by intensive clinical study and treatment through an adequate staff of psychiatrists, internists and nurses, with ample facilities and complete equipment for the purpose.

"3. Scientific research into the nature, causes and results of mental abnormality in the hope that greater knowledge may discover more effective methods of prevention and treatment.

"4. The adequate teaching of psychiatry in medical schools associated with mental hospitals where, as stu-

dents in medicine, the future family physician and general practitioner, who have almost exclusive opportunity in the recognition and treatment of incipient mental disease and mental defect, might learn to foresee and detect the earliest mental symptoms and institute at once preventive and remedial measures.

"5. Establishment of mental clinics, available: (a) To mental patients who might seek advice and treatment while able to live at home and, perhaps, continue their work. (b) To their families and friends who need instruction as to the nature and probable outcome of mental affection and guidance to proper agencies of relief of situations which might involve harm to the patient and danger to the public. (c) To educators, juvenile courts, and charitable agencies whose first effort should attempt to exclude or confirm existence of mental defect or mental disease as causative of backwardness in school, of delinquency and crime, of inefficiency and dependency.

"Such service could best be rendered through the outpatient department of a mental hospital whose social service arm could reach the community, its facilities for exhaustive study and treatment be accessible to special patients, and its *Preventorium* receive borderline mental patients for preliminary observation, prevention and treatment.

"The foregoing are the paramount aims of institutional provision as expressed in the Psychiatric Hospital idea, which may be epitomized thus: the prevention of mental abnormality; its cure or amelioration; scientific research into its nature, causes and results; diffusion of such knowledge through adequate teaching of psychiatry in medical schools to facilitate early treatment, and, through mental clinics, to serve the mentally affected within the community, in the schools, before the courts, or in need of charity. Other aims of institutional provision are:

"6. Adjustment of conditions of living, occupation and diversion to individual need, particularly of long residence patients, so that they may be as nearly normal as mental infirmity permits, and render such unfortunates happy and useful within their limitations.

"This would be the realization of the *colony* idea, whose main purpose is home life with natural interests

and duties, in family groups with separate environment of varying character and extent, appropriate to social, occupational and diversional requirements and personal peculiarities, on country estates under the medical supervision of an associated psychiatric hospital in the city.

"7. Humane care and ministration to the infirm and other patients with dangerous and degenerate tendencies who require medical and nursing attention and insight into their mental states, in order that they may be properly tended, safeguarded, and relieved of asperities in their situation as much as possible by kindness and occupational diversion.

"Such duties are best discharged in *infirmaries*, which may be at some distance in the country, but equipped with a competent medical staff and the best facilities for treatment, under supervision of the psychiatric hospital management.

"8. Segregation as a protection of the mentally affected who are weak and neglected and as a defense of the public against present dangers in the community and future menace by reproduction of their kind."

**The Curatelle and Modern Psychiatry.** George W. Jacoby<sup>3</sup> uses the amended German law as a basis for his discussion. He says:

"To all kinds of curatelle, the following points are common: The curatelle is applicable not only to persons of full age, but also to minors more than 7 years old. A curator must be provided forthwith for every incompetent.

"So far as control of the person of the ward is concerned, the law establishes a difference between an incompetent minor and one who has attained his majority. Over the former the guardian possesses general powers of discipline, while over the latter he may exert his authority only so far as may be requisite in connection with the special failing for which the curatelle has been instituted. While a curatelle on account of insanity deprives the incompetent of all legal personal will, the curatelle on account of feeble-mindedness, improvidence, or inebriety is not so far reaching, in that the incompetent is placed upon the same plane as a minor more

(3) New York Med. Jour., May 20, 27, and June 3, 1916.

than 7 years of age, and may carry out all legal acts in the same way as such a minor. He can, with legal validity and without the coöperation of his curator, carry out property transactions so long as they accrue to his benefit or profit, while, on the other hand, all such transactions which do not accrue to his benefit are binding only if they have been effected with the sanction of his curator.

"In one particular, however, the incompetent, so declared on account of insanity, differs from a minor more than 7 years of age. While the latter, when not declared incompetent, and still under the authority of his parents or of an ordinary guardian, can freely make effective testamentary disposal of his personal property, provided that he has completed his sixteenth or eighteenth year, as the case may be, an incompetent, whether so declared on account of insanity, feeble-mindedness, improvidence, or inebriety, can make no valid testamentary disposal so long as the curatelle remains in force."

The author considers the various forms or kinds of curatelle as follows:

*"Curatelle on account of insanity.* Continuing with the German statute as a basis, we find the code of civil procedure states, 'a person can be placed under guardianship who, as a result of insanity, is unable to conduct his affairs.' But what are we to understand by 'insanity'?

"The definition given by this code of civil procedure is based on that found in the code of criminal procedure. The latter defines insanity which renders a person irresponsible in the following way: 'A punishable action does not exist if, at the time of the commission of the act, the offender was in a state of unconsciousness or in a condition of disordered mental activity due to disease, in consequence of which his free determination was impossible.'

"But the existence of insanity alone is not sufficient ground for the institution of a curatelle, for the law expressly maintains that the person to be declared incompetent must, in consequence of insanity, be incapable of transacting his affairs. It would seem, therefore, that the notion of insanity necessary or sufficient for the in-

stitution of a curatelle and the notion of insanity in general, are not one and the same.

"A special class of insane persons, who often can not be legally declared insane, but who at least require the protection of guardianship, consists of those known as querulants or litigants. Every psychiatrist has had to deal with litigious insane persons who have sacrificed their entire fortunes in order to enforce their supposed rights. In these cases, when a curatelle on account of insanity can not be obtained on account of legal difficulties, the establishment of guardianship on account of extravagance would meet all requirements.

"Under certain conditions, therefore, more than mere proof of the existence of insanity is demanded for the appointment of a guardian. On the other hand, in some ways the law demands rather less for the institution of a curatelle than it does for other purposes, for it is not necessary that a disease be present that will abolish all freedom of the will, but only a defect that disables the patient from conducting his affairs.

"When we say the person to be placed under curatelle must be incapable of transacting his affairs, we must understand 'affairs' in its broadest sense and not by any means limit the word to transactions relating to property rights. The patient, for example, may be placed under curatelle because he is unable to transact his financial affairs properly, or because he is unable to manage his household, or because he cannot carry on his profession or trade. It is not sufficient, however, that he be unable, in consequence of disease, to carry on certain branches or parts of his affairs, while, on the other hand, he is not required to be equal to managing all affairs that devolve upon him; the question is, rather, whether he is incapable or incompetent in any particular which may be decisive for the totality of his personal relations or conditions."

The entire question of the relation of the mental condition to business competency can be placed in its proper light only by considering it from two points of view. On the one hand, a person may be competent to transact his affairs notwithstanding the existence of a pronounced

psychosis and need therefore not be placed under curatelle, while, on the other hand, a person may be incompetent to transact his affairs, although he be not suffering from any form of actual insanity, and therefore must be placed under curatelle.

A question which is of direct medical interest, however, is whether the law has not provided some temporary means for protecting an alleged incompetent during the period that his competency is being considered by the court.

In the main, says Jacoby, the existing special provisions of the law relating to curatelle do not furnish adequate protection against the dangers of this unbridged interval. For that reason, the law has found it necessary to provide an effective general means of protection for this critical period, which varies in form according to whether we are dealing with persons who have attained their majority or with minors still under parental authority. A person who has attained his majority and in whose interest curatelle proceedings have been instituted may be placed "under temporary guardianship" if the court considers that procedure necessary for the protection of his person or his property. On the other hand, minors who are under parental authority, but for whom no curatelle has been instituted as yet, may receive a guardian if the premises seem to warrant the ultimate institution of a guardianship in consequence of insanity.

When the curatelle proceeding has been brought before the court, it is the duty of the tribunal to undertake an investigation of the mental condition of the person for whom the curatelle is desired, basing its inquiry on the evidence and facts enumerated in the petition. First, however, the supposed incompetent must be given opportunity to adduce facts which he may consider of importance in opposition to the application. Witnesses or experts are subpoenaed and examined by the court and, in such a procedure, they are under the same obligation to appear and to testify as in all civil actions at law. Whatever the testimony, a decision can not be given until the subject of the inquiry has appeared in person and has been examined in the presence and with the aid

of one or more experts, even if coercive measures must be employed to bring about this examination.

In certain cases the experts, notwithstanding, a careful examination of all the evidence, will not be able to give a conclusive opinion until after a prolonged observation of the subject of the inquiry. In such cases the court may—but only with the consent of the applicant for the curatelle—order the patient transferred for observation to some institution or asylum.

That a transfer to an asylum can be effected only with the acquiescence of the applicant for the guardianship, has already been stated.

“Finally, the decision for transfer to an institution must contain a statement of the length of time the internment should last, the longest permissible period of confinement for the purpose of observation being six weeks.

“Before such internment can be decided upon, the relatives and others who are responsible for the care of the person to be interned, as well as the district attorney, are to be heard. When the decision for a transfer to an institution for the purpose of observation has been reached, a notice of the decree is to be served upon the person to be placed under guardianship and upon the legal representative in charge of his person, if such there be. The decision, however, is not final, for either the person to be placed under guardianship or his legal representative may, within two weeks, appeal to a higher court.

“The court, in reaching a decision, has only the alternatives, to refuse the guardianship or to grant it. If the guardianship is refused the decision is to be officially communicated to the proponent, the district attorney, and to the person for whom the curatelle has been sought. The right of appeal against the decision exists for the proponent and for the district attorney for a period of two weeks. In case of appeal, a decision is obtained from a higher court, and then the right of appeal to a still higher tribunal may be invoked.

“If, on the other hand, the curatelle is decreed, notice of the decision need not be given to the person placed under guardianship, but must be served officially upon

the proponent, upon the district attorney, and furthermore, if the sufferer is still under parental authority, upon the legal representative in charge of his person. Finally, the decision must be placed before the court of chancery.

"As a direct result of this decision, the person placed under curatelle receives a guardian whose province it is to represent him legally and to care in all ways for his person and his property. The appointment of the guardian is made officially by the court of chancery.

"But we must consider the question what to do, if after institution of the curatelle actual recovery, or at any rate a marked improvement takes place, or if one of the persons in authority alleges, justly or unjustly, that such improvement has taken place. A change in the legal status of the incompetent can occur only if the court sets aside the curatelle. The decision to annul a guardianship can be the result only of a formal application for its termination, and the applicant may be the restricted person himself, his legal representative, or the district attorney. The procedure for annulment corresponds in its details to that for the institution of the curatelle. The application will be granted only if the proof submitted convinces the court that the reason for the existence of the guardianship has disappeared—that is, that the mental condition of the declared incompetent has improved to such an extent that he is again capable of transacting his own affairs."

"In employing the term insanity, decisive alone in the eye of the law is the fact that the person suffering from a mental defect is unable properly to manage his affairs, and that at the same time, in order to afford adequate care and protection, it is necessary to place the incompetent upon a *par* with a child *less* than 7 years of age. It may well happen, however, in accordance with the nature, intensity, and course of the disease, and the pecuniary occupational, and other social condition of the patient, that sufficient care and protection will result through treating the patient as a minor *more* than 7 years of age.

"For the latter class of cases the curatelle on account of feeble-mindedness has been established, because in no

event should a person's freedom of action be more restricted than the necessities of the particular case may require. In other words, whether a curatelle should be enforced on account of insanity or on account of feeble-mindedness depends not upon the nature and degree of the mental defect, but upon the question whether adequate protection is obtained through enforcing the legal status of a minor *more*, or one *less* than 7 years of age.

"The procedure for the establishment of a curatelle on account of feeble-mindedness and those for appeal and annulment are, with but slight differences, the same as those for insanity. Through the curatelle on account of feeble-mindedness, as we have said, the incompetent is placed upon the same plane as a minor of more than 7 years of age. The curatelle on account of feeble-mindedness, like that on account of insanity, may be annulled when the cause for its existence no longer obtains; *i. e.*, when the incompetent has so far improved as to be able again to manage his affairs. But what if a patient has been legally declared incompetent on account of feeble-mindedness, and subsequent events show the curatelle on account of feeble-mindedness to be insufficient to meet the object sought? Such may well be the case if conditions for one reason or another have been misunderstood, if a patient's mental state has changed, or if his responsibilities have materially altered. Thus a patient having some simple occupation or social relation which he was perfectly able to master, may through inheritance, be forced into a higher and more complicated one. In such an instance nothing would preclude a curatelle on account of feeble-mindedness being changed, of course upon application, into a curatelle for insanity."

Very often, says Jacoby, extravagance is a dominant trait of congenital feeble-mindedness. Consequently, if in the individual case it is possible to demonstrate the existence of a psychic disorder which prevents the patient from managing his affairs, it will be easy, either on the ground of insanity or of feeble-mindedness, to obtain a curatelle that will protect the incompetent and his family against the results of his extravagance. But if such protection is the sole object, a curatelle on the ground of insanity or feeble-mindedness is not essential;

for according to the law, extravagance in itself, without relation to the underlying causes, is under certain premises a ground for curatelle. Furthermore, as the law views it, there is no extravagance when, according to certain doctrines, the dissipation of values, time, and working capacity is brought about as a result of neglect or omission. In other words, only that which affects tangible properties is legally an extravagance. That the spendthrift who has been declared incompetent—just as the one declared incompetent on account of insanity or on account of inebriety—is, in the eyes of the civil law, on a *par* with a minor not more than 7 years of age, and that in addition he is incompetent to make testamentary disposal of his belongings, has already been explained.

“Proceedings for the establishment of a curatelle on account of extravagance vary only slightly from those necessary for a curatelle on account of feeble-mindedness. The differences are legal ones which concern the physician little, but what may be of interest to him is that the court can not, of its own initiative, order the production of a medical certificate before the beginning of the inquisition, as it may in the proceedings for a curatelle for other causes.”

The decision for the establishment of this form of curatelle may be given without personal examination of the spendthrift and without presentation of medical expert opinion. Nor is an order for observation in an institution admissible. Jacoby states that as the parties making the application have the right to offer all proper means of proof, they can not, however, be prevented from adducing medical expert opinion and calling medical experts.

“He may be placed under curatelle who, in consequence of inebriety, is unable to manage his affairs or exposes himself or his family to want or endangers the safety of others. . . .”

What has been said in regard to the pathologic aspect of extravagance applies with equal force to inebriety.

A drinker can not be placed under curatelle solely because he is addicted to the exclusive use of alcoholic beverages, but certain contingencies which the law speci-

fies must have arisen. These are three, any one of which suffices: The first, which is also a presumption for the establishment of a curatelle on account of feeble-mindedness, is an inability to manage one's affairs; the second, which coincides with the presumption for the establishment of a curatelle on account of extravagance, is the danger of want; and the third is the endangerment of the safety of others.

The "incompetent" inebriate is legally on a *par* with a minor over 7 years of age. The guardian has the right and must assume the duty of caring not only for the property of the inebriate, but also for his person unrestrictedly in the case of minor inebriates, and so far as is called for by the purpose of the curatelle in the case of major inebriates. The guardian can and must determine the place of sojourn of his inebriate ward. It need scarcely be stated that in many instances the first step under a guardianship would be the internment of the patient in an asylum for inebriates.

Jacoby concludes by directing attention to certain main points in which our curatelle laws require reform, and in which, as is shown by the German law of curatelle, they are capable of being improved. These are:

"1. Agreement in regard to the fundamental views about the psychic states which necessitate the institution of a curatelle, namely, insanity, feeble-mindedness, improvidence, and inebriety.

"2. An extension of the interpretation of the term, inebriety, so as to apply to the habitual use of narcotics or other habit-forming drugs, so that it might be possible to place under guardianship, not only alcoholic inebriates, but also inveterate users of morphine, cocaine, or other drugs, and thereby permit all such unfortunates to be forcibly subjected to treatment.

"3. Adoption of the concept, hitherto not accepted by Anglo-Saxon law, of restricted responsibility, through which it would be possible to place a person under guardianship without stigmatizing him as insane, or to refrain from placing a psychically defective individual under curatelle, if his capability to transact business properly is not affected by his psychic deficiency.

"4. Appointment of the medical expert by the court

in order to guarantee an absolutely impartial opinion."

**The Causation and Cure of Certain Forms of Lunacy.** Rupert Farrant<sup>4</sup> publishes a summary of work directed to the causation and cure of lunacy, carried on during the last seven years.

The work consists of the microscopic examination of sections taken from the pineal, pituitary, thyroid and sexual glands—first, at different ages and periods of life such as puberty, menopause, and childbirth; secondly, the effect induced in these glands by the acute and chronic toxemias; and thirdly, the changes found in cases of lunacy.

From some 3,000 sections it is found that these glands vary at different ages and periods of life; with advance of life they tend to atrophy.

It is found that the pineal gland reacts to certain toxemias, the ultimate result of which is fibrosis. The pituitary reacts in a similar manner, the terminal result of which is fibrosis; intermediate stages of hyperactivity are seen, and the formation of cysts and adenomata. The reaction of the thyroid to certain toxemias, with the induction of hypertrophy, cysts, and adenomata, Farrant has already described in his lectures as Hunterian Professor in 1915.

In primary and secondary amentia atrophy of the pineal, pituitary, and thyroid were found in three main groups of cases. In dementia praecox an alteration was found in the glands which varied with the duration of the case. Alteration and degeneration were also found in other cases of dementia. In some cases of acute confusional mania, melancholia, manic-depressive and other forms of insanity, changes were found in the thyroid, pituitary, and sexual glands. The changes varied from hypertrophy to atrophy.

Clinical examinations were made of some 1,000 cases of insanity, analogous to those from which the pathologic sections had been prepared, for signs of enlargement or atrophy, and the presence of excess or deficiency of secretion from these glands.

The thyroid gland was frequently found to be abnormal in children, adolescent and adult lunatics; its size

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(4) Brit. Med. Jour., June, 1916.

varied from considerable enlargement to complete atrophy, and the general condition from one of hyperthyroidism and exophthalmic goiter to myxedema and cretinism.

The pituitary gland was found sometimes to have given rise to symptoms of hyperpituitarism and to apituitarism, in idiocy, dementia praecox, and other forms of insanity. Enlargement and atrophy of the pituitary were deduced from x-ray photographs of the sella turcica and clinoid processes.

Signs of alteration in the pineal were found, especially in children and adolescents, with consequent symptoms of hyperpinealism and apinealism.

Alteration was found in the sizes of the testicles associated with ductless gland changes. Testicular atrophy was well seen in cases of apituitarism. From the histories of cases it was deduced that the stimulation of the sexual organs or its sudden cessation was associated with altered mentality varying from mania to exhaustion psychosis; altered mentality was also found in cases of double castration. Hypertrophy of the prostate was often found to be associated with altered mentality, and after complete removal cases were frequently associated with depression or melancholia.

It is deduced that many cases of lunacy may be classified according to the toxemia present and the change that it has induced in the ductless glands.

The effect of the toxemia varies with the intensity, duration, and the age of the patient; the first effect is stimulation, the final fibrosis. In the fetus and in childhood an acute or chronic toxemia may induce atrophy of a ductless gland, and consequent maldevelopment and idiocy. Three groups stand out when the pituitary, pineal, or thyroid is atrophied. Cases of dementia praecox exhibit a polyglandular syndrome, and different gland types are to be found, and the appearance of the cases varies whether the gland is hypertrophic or atrophic.

In adults certain toxemias react on these glands, especially the thyroid and pituitary, and induce stimulation, hypertrophy, and, finally, atrophy. The alteration in the amount of secretion—whether excess, deficiency, or

absence—induces an altered mental state, and this, combined with the effect of the toxemia, renders the patient insane or liable to insanity from slight mental stress.

Alteration in the sexual glands, whether primary or secondary, leads to altered mentality up to insanity.

**Mental Disturbance in Soldiers.** According to Urstein's<sup>5</sup> observation, psychic disorders caused by brain trauma develop usually only when more or less extensive portions of the gray matter of the cortex are involved, though it is possible for even circumscribed lesions of the brain as, for instance, fracture of the skull, hemorrhages, etc., to affect the mind. The immediate result of brain trauma is mental confusion. The patients appear somnolent, dazed, forgetful and absent-minded. In severe cases unconsciousness may last for hours and even days. Other symptoms are headache, fainting, dizziness, vomiting, slow pulse, pupil disturbances, paralysis and convulsions. The mental confusion usually shows immediately after brain trauma, but in some cases only after an interval of hours or days. In addition there are observed changes in the character; excessive sensitiveness, excitability and irritability, exhilaration, maniacal states and hypochondriac ideas. In general, the clinical picture resembles that of traumatic delirium. The symptoms of delirium become more pronounced when the course is unfavorable, which may be due to abscess formation or to a meningo-encephalitis. In such cases somnolence sets in, followed by coma, convulsions, paralysis and rise in temperature.

The most frequently observed psychosis was catatonia, next in frequency, psychopathic constitutional anomalies, epileptic insanity and finally manic-depressive states. Urstein has also encountered cases of so-called exhaustion psychoses, hysterical psychoses and progressive paralysis of the insane. The latter was observed only in soldiers over 30 years old. There does not seem to be any specific psychoses, according to Urstein. He claims that a psychosis develops only when there is a certain predisposition. That is, the elements of the psychosis were present before the man

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(5) *Russkiy Vrach.*, vol. 15, No. 11.

went to war; the latter but hastened its development. In general, the so-called war psychoses do not differ from those in time of peace though the clinical symptoms may be somewhat peculiar depending on the character of the warfare.

**Traumatic War Neuro-Psychoses.** Gerver<sup>6</sup> divided the traumatic lesions of the nervous and psychic spheres (in soldiers) into the traumatic psychoneuroses and traumatic neuroses. Both may be traced to contusions, especially those caused by air concussions or by wounds. The clinical picture of the traumatic psychoneuroses resembles that of common traumatic neuroses, differing from the latter by peculiar hallucinations, illusions, obstinate focal symptoms and a peculiar condition of the upper lids, an inability to raise the upper lid after it has remained closed for a few seconds. The traumatic psychoneuroses from air contusion are especially severe, as they show signs of diffuse lesions of the central nervous system. In those caused by wounds, the local symptoms predominate and the course is milder. The air concussion causes probably diffuse pathologic anatomic changes in the central nervous system, such as molecular changes, miliary hemorrhages, thrombosis, and necrosis. The treatment must be individualized, rest being the most important item.

**Prolonged Pharmacologic Hypnosis and Its Application for the Cure of Some Psychoses.** According to G. Epifanio,<sup>7</sup> pharmacologic hypnosis consists in the administration of an hypnotic for several days in succession in doses so regulated as to induce a more or less continuous sleep. The term "hypnosis" is preferred by Epifanio to "narcosis," which implies abolition of sensibility and complete dulling of the sensorium, whereas in hypnosis consciousness can be awakened and the general sensibility is almost intact. In psychoses due to infection, in febrile delirium, and delirium tremens, a profound sleep often marks the end of the mental disturbance. The writer accordingly thought of provoking artificially a sleep of long dura-

(6) Russkiy Vrach., Oct. 3, 1915.

(7) Riv. di patol. nerv. e. ment., 1915, vol. 20, p. 273.

tion as a means of curing some forms of mental disease.

He records ten cases in which he administered subcutaneous injections of luminal-sodium in doses of from 20 to 80 grammes for from five to twelve days in succession. His results were as follows: During the treatment and the following days there was an increase in body weight. Permanent recovery maintained up to the time of writing, *i. e.*, for about two years, occurred in (1) a form of dementia praecox with severe but early symptoms; (2) in an undetermined case resembling in some respects dementia praecox and in others manic-depressive psychosis; (3) a case of pure mania in a young woman; (4) a case of manic-depressive psychosis.

No improvement occurred in a case of old hebephrenia and in a cataleptic and stuporous case with advanced dementia. In a very severe catatonic form resolution of the catatonia took place, but dementia persisted. In a grave degenerative form in an individual with a bad family history there was only slight improvement.

Epifanio discusses the action of a prolonged sleep on consciousness, memory, emotions, ideation, volition, etc. The most important result was the disappearance of hallucinations. No bad results occurred from the use of the drug.

**Rôle of Hallucinations in the Psychoses Based on a Statistical Study of 514 Cases.** Forrest M. Harrison<sup>8</sup> concludes:

1. Hallucinations are among the commonest of symptoms met with in the insane, occurring in approximately 40 per cent. of the cases.

2. Of the various types, those of hearing are most frequent, these occurring either separately or combined in 90 per cent. of the cases hallucinated. Next in frequency are those of hearing and sight combined, and then come visual disturbances alone.

3. The content of the hallucinatory percepts were not characteristic for any particular psychosis.

(8) Jour. Nerv. and Ment. Dis., March, 1916.

4. Visual disturbances seem especially peculiar to the catatonic praecox group.

5. Hallucinations are common in dementia praecox, occurring in practically all the cases. On the other hand, they are rare in the manic-depressive group, seldom if ever occurring typically. This fact is of diagnostic importance.

6. Hallucinations are rare in arteriosclerotic dementia and senile dementia, occurring in approximately 20 per cent. of the cases.

7. Hallucinations are rare in sane persons, even though they be of a psychopathic make-up.

**Investigation of Adrenaline Mydriasis in Normal and Insane Persons.** W. M. von der Scheer<sup>2</sup> states that adrenaline mydriasis occurs slightly more frequently in the various psychoses than in normal persons. It is at times very marked in the insane, and lasts longer particularly in catatonia and epilepsy. Adrenaline mydriasis has no practical value in either the diagnosis of dementia praecox or the differential diagnosis between the functional and organic psychosis.

**Pathologic Findings in the Sympathetic Nervous System in the Psychoses.** A. Myerson<sup>1</sup> calls attention to the fact that compared with what has been done by the neuropathologists on the central nervous system, that done on the sympathetic sinks into significance. From a study of the material obtained from fifty consecutive autopsies, he states:

First, the semilunar ganglion is apparently often acutely injured in general infections and in enteritis, as is shown by the presence of the axonal reaction and other changes.

Second, the semilunar ganglion is the seat of degenerative processes to an extent probably greater than cord, brain or Gasserian ganglion. The degenerative changes are here designated under the general term neurothrepsia (Levaditti). It is probable that these changes represent an early and marked senility, and in this Myerson states that he is in accord with Lugaro.

Third, there is a decided absence of marked reac-

(9) Neurol. Centralbl., September, 1915.

(1) Amer. Jour. Insan., April, 1916.

tive change (lymphocytes, plasma cells, etc.), such as are prominent in the central nervous system, Gasserian ganglion, and the related organ or adrenal. Even in general paresis these are absent.

Fourth, there is a curious, though not prominent, increase of eosinophilic connective tissue cells which in a case of bovine tuberculous enteritis cited by the writer seemed to have a phagocytic attraction for injured nerve cells.

It is to be emphasized that the above findings apply, in Myerson's experience, only to the psychoses.

*Conclusions:* Two prominent conclusions, he states, stand out as worthy of emphasis.

First, the semilunar and other sympathetic ganglia and the autonomic ganglia merit the close attention of pathologists. The part the vascular and glandular system under their control plays in all the great vital processes as well as in the creation and modification of the emotions, indicates that a mere comprehensive and systematic study may throw light on the problems of old age as well as on the psychoses. Regarding the latter it may be stated that quantitatively greater changes in the cases of insane people may well have a value equivalent to qualitative changes, since in many of the psychoses the evolution of the disease is from temperament to insanity.

Second, the interpretation of morbid phenomena needs to take into account the presence of nerve cells in the organs, such as the aorta, the heart, the intestines, stomach, genitalia, etc. Symptoms may well arise because of injury to these peripheral cells either as an antecedent, or as a consequence, of the disease process. We have, as recent experiments show, drugs that have a peculiar and selective power on the nerve cells of the autonomic and sympathetic systems. These should be experimentally as well as therapeutically used in conditions in which the symptoms are even in part vasomotor and glandular.

**Treatment of Depressed States with Sedobrol and Bromides.** A. Ulrich<sup>2</sup> details the technique of bromide treatment in melancholia and says that continued bro-

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(2) Cor-bl. f. schweiz. aertze., January, 1916.

mide treatment is successful providing it is carried to the point of producing bromine intoxication. A change of the depressed state of mind into a euphoric one which later becomes normal, follows the treatment.

By "*Bromrausch*," *Bromarkose*, bromism, is understood the condition of cerebral bromine intoxication, manifesting itself by uncertainty in walking or staggering with ataxic gait, characteristic speech, and writing and memory disturbances.

The *Bromrausch* can be continued in most cases until a visible change in mood occurs, as in euphoria with peculiar flow of ideas; the patients becoming good natured but unable to express themselves adequately. In older patients and in cases of long duration we do not find the lasting euphoria at the highest point of the *Bromrausch* but later, and it is advisable to reduce the dose gradually when the latter point has been reached.

If after a *Bromrausch* the change in mood is not lasting or sufficiently great, the treatment is to be continued through several phases. Several bromine intoxications can be produced successfully without interrupting the treatment.

In decrepit or arteriosclerotic patients, who in the course of treatment show marasmus the continuation of the treatment is contra-indicated.

In employing this method Ulrich removes sodium chloride from the food and replaces it with bromide. In short and intensive courses he uses this substitution not only in soup but bread and vegetables also. The duration of the bromide administration is to be regulated by individual cases. Strong persons can tolerate a short and more intensive treatment, whereas older and less rugged persons are to be treated by a slower procedure.

In the first group from 4 to 5 gm. of sodium bromide (4 to 5 tablets of sedobrol) can be given. In the other cases he begins with 2, finally 3 gm. of sodium bromide (2 to 3 sedobrol tablets) and slowly increases the dose.

When the bromide intoxication is developed, then the bromide is slowly diminished and from 3 to 5 gm. of sodium chloride is added daily to the food. Under no condition must the bromide be discontinued suddenly and permanently. When reducing the bromide one must

be guided by the state of emotion and sleep in the patient. If there is depression and crying reappears, or if sleep is insufficient, it is advisable to increase the bromide temporarily. What is generally called bromism, *i. e.*, acne pustules of the face and body, has nothing to do with cerebrospinal bromism. If the acne is to be avoided in persons whose skin is sensitive, Ulrich advises the administration of a few drops of Fowler's solution, twice daily after meals.

#### ALCOHOLISM.

**Alcoholism a Symptom.** William A. White<sup>3</sup> calls attention to a series of mental conditions in which alcohol rather characteristically enters, at least in certain individual cases. Alcohol quite characteristically enters into the picture, not only in alcoholic psychoses so-called, but in manic-depressive psychoses, the dementia praecox group of psychoses and, of course in the early stages, particularly paresis. He particularly calls attention to this psychosis, which consists, to put it briefly, in alternating periods of depression and exaltation, interrupted or followed by a normal interval of variable length. Occasionally, a patient suffering from this psychosis shows the initial symptoms of his excitement by over-indulgence in alcohol, and it is sometimes exceedingly difficult to differentiate the condition from one purely the result of alcoholic indulgence. The individual who has become alcoholic contemporaneously with his attack of excitement ceases his indulgence when he again becomes normal and has no tendency to drink again until the excitement recurs. Such evidence seems to White to be very strongly suggestive of the erroneous of the conception of alcohol as a habit-producing drug.

Inebriety, in White's opinion, must be considered as a neurosis, and from this point of view has the two fundamental traits of a neurosis which are of prime importance in explaining its symptoms.

The feeling of inefficiency and flight from reality, the ear-marks of a neurosis, are the ear-marks of alcoholism and now we can understand why alcohol has been called

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(3) Interstate Med. Jour., June, 1916.

a stimulant, and why it has been called a habit-producing drug. It has been called a stimulant, because the individual, who is incapable of facing reality and has had to take alcohol to escape, has had also the best possible reason for taking it—namely, that it would help him to meet reality.

**Alcohol and Heredity.** Charles R. Stockard<sup>4</sup> reports his experiments on "The Hereditary Transmission of Degeneracy and Deformities by the Descendants of Alcoholized Mammals." He concludes:

*The data from alcoholized male lines indicates that the female offspring from alcoholic males are less viable and more frequently deformed than the male offspring. And heterogeneous matings of such male and female offspring further emphasize the same inferiority on the part of the female offspring from treated males.*

The data from alcoholic female lines indicates that the male offspring from alcoholic females are inferior in quality to the female offspring. Heterogeneous matings of such male and female offspring further prove the inferiority on the part of the male offspring from treated mothers.

Finally, the experiments show the hereditary transmission through several generations of conditions resulting from an artificially induced change in the germ cells of one generation by treatment with alcohol. And they furnish data of importance bearing on the pathologic behavior of the carriers of heredity as well as the differences in behavior between the two types of germ cells produced by an animal carrying heteromorphic chromosomes.

**Alcoholism and Feeble-mindedness.** Henry H. Goddard<sup>5</sup> in a discussion of alcoholism and feeble-mindedness, discusses Stockard's results and says:

"The experiments are of extreme importance for the whole problem of heredity and the transmission of acquired conditions. But in the interest of truth the tendency that one may have to draw conclusions as to the offspring of alcoholized human beings must be carefully restricted. In the first place, it must be

(4) Interstate Med. Jour., June, 1916.

(5) Interstate Med. Jour., June, 1916.

remembered that we have no way to measure the relative strength of the injury. There may be all the difference in the world between subjecting a guinea-pig to an atmosphere of alcoholic fumes and subjecting a man to the most frequent doses of beverage containing a high percentage of alcohol. Secondly, it must be remembered that it is unsafe to draw conclusions of the effect of a drug upon man from its known effect upon the lower animals. And lastly, it must be remembered that Stockard's results were largely in the form of physical deformities. While he cites one case in which the brain appeared quite different in some respects from a normal brain, nothing is definitely shown as to the effect of this upon the mentality of the guinea-pig, or the frequency of this kind of deformity."

In connection with his study of the 300 cases at Vineland which has already been published under the title of "Feeble-Mindedness—Its Cause and Consequences," Goddard has accumulated many facts on the subject of the alcoholic or non-alcoholic conditions of the parents of the children in the Vineland school.

The 300 children whose families were investigated had 11,389 relatives, or an average of a little less than forty to each family. This means that on an average he was able to chart for each family represented by a child in this school forty people including the parents, grandparents, cousins, uncles, aunts, etc. Of the 11,389 persons, 365 were alcoholic, that is 3.2 per cent. Alcoholic here means drunkard.

In conclusion he says that while his results are mostly negative and "I can simply say that we have failed to show that alcoholism of the parents causes feeble-mindedness in the children," the results could be made positive if it were safe to argue that were there a casual relation between these two things. It ought to be discovered in a mass of data such as that which he has considered.

His belief is that feeble-mindedness is pretty well accounted for by heredity, neuropathic ancestry and accidents (including diseases in the child and in the mother); with the possibility that given these condi-

tions the addition of alcohol may in some cases throw the case into the feeble-minded group where without the alcohol it would have escaped.

## CATATONIA.

**Focal Lesions of the Cortex of the Left Angular Gyrus in Two Cases of Late Catatonia.** E. E. Southard and M. M. Canavan<sup>6</sup> report these cases to suggest a genetic relationship between the focal lesions and the subjective symptoms. They feel that they cannot offer proof of such relationship and merely hope to excite others to opposition or to similar reports. They present two cases which suggested and obtained the diagnosis "dementia praecox," although the cyst of softening in one and the solitary tubercle in the other may well be regarded as withdrawing the cases altogether from the dementia group and settling them in a group of nondescript, coarsely organic and destructive brain lesion cases.

Whether the lesions had a direct mechanical effect upon the tissues to give rise to the symptoms must remain obscure. That the gliosis with its contractile tendencies was more marked in the supratentorial region than in the infratentorial is merely an interesting fact. They leave unanswered the question, from the data of these two analogous cases, that catatonia or catatonic-form symptoms may occasionally be mechanical in origin. The authors are especially interested in the fact that the isolated lesions in these cases are in the parietal regions, a region which has been stated in previous work to be correlated with catatonic symptoms.

## DEMENTIA PRAECOX.

**Histology of Dementia Praecox.** Richard Zimmerman<sup>7</sup> says that lipoid sclerosis, fibrolysis, glial increase and ameboid glia cells are found. The cortex is deeply and severely involved by tissue change. Chronic cell and tissue changes are most prominent in cases of

(6) Amer. Jour. Insanity, January, 1916.

(7) Zeitschr. f. d. ges. Neurol. u. Psychiat., January, 1916.

believe that trauma renders the brain more susceptible to the syphilitic virus. Among these writers are Mendel (1904), Giesder (1905), and Froissart (1907).

3. Those considering trauma an occasional accessory factor. Among these are Foville, Gudden, Magnen, and Regis.

4. Those who deny the rôle of trauma altogether, such as Mercier, Kaplan, and Brissaud.

Benon promptly rules out the view held by the first two groups of writers but admits that the question of the occasional accessory rôle of trauma is still an open one though great skepticism is in order.

Clinically, the post-traumatic cases in no way differ from the ordinary ones. In their relation to the trauma, they may be classified thus:

1. Those in which the pre-existing disease is aggravated after the injury, which may be a light one.

2. Cases with rapid onset immediately after the injury.

3. Cases with onset years after an injury.

4. Cases with insidious onset and steady progression coming on soon after an injury, with headache, dizziness, tinnitus, visual disturbance, such as scotomas and insomnia as prominent symptoms.

It is only in this last class that the trauma may be looked upon as a contributing factor of some consequence.

#### PARESIS.

**Conjugal Paresis.** Commenting upon a case of conjugal paresis which he reports, H. H. Drysdale<sup>1</sup> says:

"This case presents some very interesting features. The father and mother are now hopeless invalids as the result of the ravages of syphilis. Their only daughter has inherited the disease, and no one can prophesy what her future health will be. The husband contracted syphilis two years prior to his marriage. The symptoms were slight and quickly subsided after a brief period of treatment. Eighteen years of syphilis preceded the development of paresis in his case.

"Unquestionably he infected his wife, but just when

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(1) Jour. Amer. Med. Ass'n., July 29, 1916.

no one can say. She did, however, become a paretic two years after her husband, who was syphilitic two years before they were married. It would be interesting in this connection to know if the number of years of syphilis preceding the appearance of paresis was the same in husband and wife.

"The disease in both cases was of the demential type, and at no time during my observation were there any signs of exaltation or grandeur."

According to the literature, cases of conjugal paresis are rare; but it has seemed to Drysdale that physicians whose experience has been more extensive than his must surely have encountered conditions of this sort.

**Comparison of Mental Symptoms Found in Cases of General Paresis with and Without Coarse Brain Atrophy.** E. E. Southard<sup>2</sup> reports that a group of thirty-eight general paretics whose brains were specially examined and described by him, has been divided into two groups according to whether there was not coarse evidence of brain atrophy. The cases without brain atrophy were termed "mild" and those with brain atrophy were termed "severe," although these designations are only approximations to accuracy; the groups are, however, in no sense "early" and "prolonged."

Symptomatically the two groups show several surprising concordances and a number of instructive divergences. Thus amnesia, motor restlessness, disorientation, dementia, and depression lead both series and in that order (except that allopsychic delusions stand fourth in the "mild" series and are far less common in the "severe"). Are amnesia and dementia therefore in no sense proportional to brain tissue loss?

Nineteen symptoms occurred in 20 per cent. or over of the paretic series, *viz.*, the five just mentioned, and nine others (irritability, defective judgment, psychomotor excitement, autopsychic delusions, insomnia, aphasia, hallucinations of doubtful or unspecified nature, convulsions, visual hallucinations) not always in like proportion in the two series. Five other symptoms occurred in each series, but symptoms quite sundered from one another in general significance.

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(2) Jour. Nerv. and Ment. Dis., March, 1916.

The "mild" cases showed a group of symptoms which might be termed contra-environmental, *viz.*, allopsychic delusions, sicchasia (refusal of food), resistiveness, violence, destructiveness.

The "severe" cases showed a group of symptoms of a quite different order, affecting personality, either to a ruin of its mechanisms in confusion and incoherence, or to the mental quietus involved in euphoria, exaltation, or expansiveness.

Some speculations are offered as to the perversion or inhibition or incoördination of inhibition which the largely irritative lesions of the "mild" cases are presumably effecting in the perhaps more seriously affected frontal areas. When these are still more gravely affected, as to the point of atrophy, then the intrapsychic disorder might well become more manifest, *e. g.*, in the distinctive symptoms of the "severe" group just mentioned.

In a series of 17,000 clinical cases (of all sorts of mental disease, alive and dead, recovered and impaired) symptomatologically analyzed, there were but ten symptoms occurring in 20 per cent. or over; these were in order, psychomotor excitement, allopsychic delusions, dementia, auditory hallucinations, motor restlessness, depression, autopsychic delusions, insomnia, incoherence, amnesia. Each of these is represented high in general paresis (*i. e.*, in 20 per cent. or over) except that auditory hallucinations are infrequent in both "mild" and "severe" cases and allopsychic delusions are infrequent in "severe" cases. There may be topographical reasons for the paucity of auditory hallucinations in general paresis. The method of production of allopsychic delusions in general paresis should be studied, since there can be no such alliance of allopsychic delusions and auditory hallucinations therein as is perhaps the rule in dementia praecox.

If we consider the next nine symptoms in order in 17,000 cases of mental disease at large, *viz.*, violence, visual hallucinations, irritability, defective judgment, disorientation, destructiveness, confusion, resistiveness, and somatic delusions, we find only the last, *viz.*, somatic delusions, not represented in either group in fair proportion, although (as above stated) confusion is poorly rep-

resented in the "mild" cases and violence, destructiveness, and resistiveness are poorly represented in the "severe" cases.

Aphasia, hallucinations of doubtful or unspecified nature, and convulsions appear to be frequent symptoms in general paresis that do not figure at all so largely in mental disease as a whole. Besides these, sicchasia of the "mild" group and euphoria, exaltation, and expansiveness of the "severe" group appear to stand out for general paresis against mental disease as a whole.

The most positive results of this orienting study appear to be the unlikelihood of euphoria and allied symptoms in the "mild" or non-atrophic cases and the unlikelihood of certain symptoms, here termed contra-environmental, in the "severe" or atrophic cases. Perhaps these statistical facts may lay a foundation for a study of the pathogenesis of these symptoms. Meantime the pathogenesis of such symptoms as amnesia and dementia can not be said to be nearer a structural resolution, as these symptoms appear to be approximately as common in the "mild" as in the "severe" groups.

#### PARAPHRENIA.

**An Evaluation of Paraphrenia.** Edward A. Strecker<sup>3</sup> says:

"Briefly, paraphrenia systematica is a chronic paranoid psychosis of late onset and extremely gradual course, in which persecutory ideas are subjected to an exhaustive mental consideration; later, grandiose delusions are engrafted and the system thus evolved is maintained without perceptible shrinkage for many years. Hallucinations, usually of hearing, appear only after the delusional condition has become well established. The dwindling of psychic life, the disappearance of personality, the loss of emotional values, catatonic phenomena, and in short all those manifestations which we regard as indicative of the *dementia* in *praecox*, are strikingly absent.

"Expansive paraphrenia presents the gradual construction of an elaborate system of grandiose delusions,

(3) New York Med. Jour., Aug. 19, 26, 1916.

with prevailing elevated effect and mild excitement. Undoubtedly many of these cases would parallel "chronic mania," a favored diagnosis ten and even five years ago. The same factors which drew the boundary line between systematic paraphrenia and dementia praecox are also present here, though they are often less clear."

The third division, confabulatory paraphrenia, is quite rare. Kraepelin has seen only twelve cases in thirty-five years. It is marked by highly elaborated and detailed falsifications of memory forming the foundation for a psychotic structure consisting largely of related delusions of persecution and grandeur.

The final variety, the fantastic form, is deserving of but little consideration as a separate disease group. Kraepelin himself recognizes its close kinship to dementia praecox. It comprises cases which show a profuse development of highly fantastic, disassociated, and ever changing delusional conceptions.

"The following factors stand out most prominently on the side of the 'disease process' theory of Kraepelin. The late onset of the psychosis, the fairly close coördination between the delusional content and the reactions of the patient, the absence of definite dementia particularly as regards the affect life, the relative freedom from disturbances of the will, and finally, the retention of personality.

"On the other hand, paraphrenia bears at least a general symptomatic resemblance to dementia praecox; in many cases the emotional field and the will are involved, even though it be only in a minor degree; the presence of a certain amount of deterioration, though this is more inclined to affect the intellectual functions and may in part be due to retrogressive physical changes, especially of the vascular system.

"The factors which seem operative in giving an unusual coloring to the clinical picture are the late age of onset with its little understood, but probably significant influence on the development of the affect and will; the possibility at least in some proportion of the cases of a manic-depressive predisposition, and, finally, the modifying effect of an inherited psychopathic constitution.

"Although at the present time the weight of evidence seems to be against the 'disease process' theory of paranoia, it is important not to minimize the value of Kraepelin's recent contribution to psychiatry. The exact observational data which he has carefully gathered and emphasized by a special designation, will be extremely useful, even though the conclusions he has drawn are generally regarded as being not entirely justified by the premises."

#### PARANOIA.

**Induced Paranoic Conditions.** Paranoid ideas induced by environment seem to be of particular interest from the fact that this condition is being separated more carefully and the chances for recovery are much more favorable than they were previously thought to be.

Arthur K. Petery<sup>4</sup> says that formerly such cases were usually brought together under the general grouping of dementia praecox and the prognosis was rated as decidedly unfavorable, but the adoption of some of the later theories regarding the causation of these conditions has led us to look at these cases in a somewhat different manner and to be more optimistic as to their outlook for recovery.

Real paranoia is a form of psychopathic personality, and is a product of necessity arising from the irritations of life, and during the life of the individual these conflict with the other elements of their existence.

The true paranoiacs are the incentives; they come in contact with persons of psychopathic natures, force their opinions on them and are accepted by them as facts, thus bringing them into the fold of the type termed "induced paranoia." The originators are usually strong and active, while the induced are weak and hysterically inclined.

These patients so long as the stimulus is present, retain these abnormal ideas and their life and actions are practically under the control of the originator; but remove the stimulus or the originator and those induced can discard the ideas either to acquire new ones or not, although during the activity of these induced ideas they are en-

(4) Med. Record, Nov. 4, 1916.

tirely irresponsible for their acts and frequently commit crimes, even murder, as a religious sacrifice. These patients often need only such an impetus to make them start to misinterpret things and to carry them out according to their own ideas or the ideas of the originator. If these persons are placed under favorable environment, free from conflicting stimuli, and are given a little assistance along the proper channels they frequently discard the ideas and return to their usual or normal condition.

Another type of induced paranoiac conditions is found in the imprisonment psychosis. This occurs, says Petery, in persons of psychopathic natures who are confined or prevented from leading the lives to which they are accustomed; they find they are unable to accommodate themselves to this state of affairs and finally to recompense themselves, develop a psychosis of degrees varying from depressions to active hallucinations. These conditions usually disappear promptly when the individuals are placed under more favorable circumstances either by removal to a hospital or by allowing them more privileges in the prison.

Another class of cases very closely allied to this type of psychosis is the psychosis of the deaf and one which seems to be becoming more prominent; at any rate we are seeing more of these cases at this time. These people are really isolated or imprisoned to a certain extent from their surroundings and being of a psychopathic makeup, with a sense of embarrassment from their affliction, find satisfaction by making explanations to themselves which they magnify until they have developed a psychosis.

Very frequently there is some pathologic condition of the ear present that causes a roaring or buzzing and which these cases are prone to interpret as "voices"; they become suspicious, people talking together are surely discussing them, and they feel that they are always the object of conversations. This usually leads to a depression; they think they are being watched; that derogatory remarks are being made about them until finally in order to escape these torments they attempt violence or self-destruction.

## MANIC-DEPRESSIVE INSANITY.

**Transient Attacks of Manic-Depressive Insanity.** Menas S. Gregory in 1908 called attention to a special group of allied manic-depressive states which he termed atypical, and which consisted of cases of very short, almost transitory duration. These patients, he then pointed out, were suffering from some form of atypical intoxication or pathologic drunkenness. In addition to their toxic symptoms, they presented the characteristic syndrome of a manic-depressive psychosis, such as typical flight of ideas, irritability, loquaciousness, and motor restlessness. In some of these cases, the manic-depressive symptoms persisted after the ordinary signs of intoxication had subsided, often within twenty-four hours, and the patient cleared up entirely.

At that time Gregory said that the mental state of these patients presented special difficulties from the standpoint of diagnosis, and that one might consider them on the one hand as the reaction of a manic-depressive personality to alcohol, or, on the other hand, as short attacks of manic-depressive psychosis complicated with alcohol.

He now says:<sup>5</sup> "After further observation I now feel that the latter viewpoint is more correct, and that these are instances of typical manic-depressive attacks in which alcoholism has been an accidental or incidental factor.

"The careful anamnesis of these patients shows that at first they drank only periodically—the so-called periodic drinkers. Although some of them still remain such, many of them have gradually become habitual and continuous imbibers as well, with episodic outbreaks of the fundamental disturbance."

"As a rule, manic-depressive patients do not suffer from alcoholic delirium. This is especially true of those with excitement. As has already been stated, most of these patients drink periodically only, and the taking of alcohol is an incident of the psychosis. A relatively small quantity of alcohol in addition to the exhilaration of the manic-depressive condition so excites the patient that he immediately attracts attention and is brought

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(5) *Med. Record*, Dec. 18, 1915.

to the hospital before he has had the opportunity of taking a sufficient quantity of alcohol, or drinking long enough to produce delirium. Even those who use alcohol in the interim between the attacks are not heavy drinkers as a rule.

In the depressed types, however, although alcoholic delirium is equally infrequent, hallucinosis is quite often encountered. In the depressed phases, excessive or prolonged taking of alcohol, likewise, does not occur, but, for an entirely different reason. While the exhilaration which is characteristic of the manic phase is lacking here, the general retardation which is the striking feature of the depressed stage prevents the ingestion of much alcohol, hence the infrequent occurrence of delirium. It may also be pointed out that some of the depressed cases may begin with an excited or mixed phase, and rapidly change into depressed types.

Transitory attacks of a manic-depressive psychosis which are not associated with alcoholism, Gregory thinks, are also very frequent, although it is difficult to demonstrate them. As has already been stated, the milder attacks pass unobserved, even by the patient himself, unless his conduct calls attention to it. Excited cases of this class are usually regarded by physicians as hysteria, or as psychic epilepsy.

Transitory depressed attacks are frequently interpreted by the patient as some common physical ailment. The motor retardation and inadequacy seem to be so much more pronounced than the purely psychic symptoms, that they attract the attention of the patient almost to the exclusion of the latter. In other words, the patient's mind is centered on the motor phenomena, so that he endeavors to find a physical counterpart for his mental disorder. If he realizes his psychic shortcomings at all, he ascribes them to the motor inadequacy, which he has converted into physical symptoms.

Gregory concludes:

"1. Short attacks of a manic-depressive psychosis, ranging in duration from a few hours to several days, are very frequent—I believe much more numerous than longer attacks requiring hospital treatment, but they are

difficult to recognize unless accentuated by some exogenous factor.

"2. These fleeting attacks are frequently marked by these very exogenous factors and are misinterpreted as mental states, due to the incidental factor, which in reality is merely accidental or incidental to an attack of a manic-depressive psychosis.

"3. Alcoholism is the most frequent exogenous agent and the great majority of periodic drinkers, especially so-called pathologic drunkards, are really examples of short attacks of manic-depressive psychosis associated with alcoholism.

"4. These fleeting attacks when mild in type and uncomplicated by alcoholism, are often mistaken for transitory mental conditions of other character, such as hysteria, psychic epilepsy, migraine, neurasthenia, etc.

"5. These short attacks of manic-depressive psychosis may be associated by the patient with disturbances of bodily functions and interpreted as common physical ailments. These conditions are frequently regarded by the general practitioner as nervous dyspepsia, nervous heart, liver and intestinal troubles, etc.

"6. The recognition of the true character of these attacks is of utmost importance from the standpoint of treatment, as misdirected therapeutics will frequently intensify the underlying condition.

"7. Important medicolegal questions frequently arise, for, during such attacks patients may commit overt acts such as assault, crimes of sexual character, attempted suicide and even homicide."

#### IDIOCY.

**Familial Mongolian Idiocy.** L. Babonneix and J. Villette<sup>6</sup> describe a family in which four daughters were affected. The other six children had died in early infancy and there had been two miscarriages. The father appeared well and normal except for leucoplakia of the cheek, the mother had died of pulmonary tuberculosis at the age of 57. Three of the sisters are described in detail and presented the following common features:

(6) *Archiv. de méd. des enfants*, September, 1916.

(a) Mongolian facies, eyes standing from without inward and from above downward and prominent cheek bones; skull brachycephalic with thick walls, hair short and scant, ears placed high and misshapen, Gothic palate.

(b) Idiocy of a special type, with extremely circumscribed mental sphere, feeble attention, incapacity for learning, sweet disposition without even occasional display of ill temper, fondness for music.

(c) Dwarfed physique, all being undersized.

(d) Certain trophic nervous symptoms, such as tendency to thinness and cyanosis of the hands, shortness and chunkiness of the feet, which are widened toward the toes. Reflexes normal.

(e) Signs of pluriglandular insufficiency, such as dryness of the hair, general muscular debility, low arterial tension.

On the negative side, the thyroid did not appear atrophic, there were no signs of hereditary syphilis, nor of rickets. General vegetative functions were in excellent condition. In all three cases there were abnormalities of the teeth not unlike the condition in cases of Hutchinson's teeth. The author suspects the possibility of congenital syphilis, but nothing is said of laboratory tests on blood and spinal fluid.

**The Spinal Fluid in Mongolian Idiocy.** H. C. Stevens' summarizes the results of his investigations as follows:

1. The Wassermann reaction on the blood serum of Mongolian idiots was positive in 33 per cent. (six out of eighteen) of the cases.

2. The Wassermann reaction on the spinal fluid was positive in 11.1 per cent. (two out of eighteen) of the cases.

3. Pleocytosis was present in no case, except in the two already mentioned in which blood was present.

4. The globulin content was increased in 100 per cent.

5. The gold chloride reaction was present in 100 per cent. of the cases.

6. The color changes of the gold chloride reaction are typical of cerebrospinal syphilis.

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(7) Jour. Amer. Med. Ass'n., April 29, 1916.

**Occupation Specialization in the Defective.** Henry M. Friedman<sup>a</sup> says that whenever we survey the defective of one kind or another who is constantly presenting himself before the physician in his professional capacity, we can not help conscientiously asking ourselves what he will do for a living after his discharge as beyond further improvement.

Instead of engaging in tiresome and always exhausting mechanical work, children should be encouraged to pursue healthy apprenticeship in previously determined suitable occupations. There would be a larger economical gain in the long run. Material would be created for enlarging many infant industries. Besides, this apprenticeship would fill the place of concrete muscular and psychologic exercise, which children would otherwise have little opportunity to get. Ultimately there would be a proper alignment of all work and of all workers. Potentially good workers would not be seeking places now filled by the unsuitable and the incompetent. Too often do we find muscular men doing work requiring little muscle. Much of their energy is going to waste unused. They should engage in occupations where they have an opportunity to use up all their available working energy. There would be less tendency for overplus in one occupation and scarcity in another. There would result a veritable social conservation of energy.

For patients having general constitutional diseases or other gross conditions, and yet who must be self-supporting, it is quite necessary, says Friedman, to advise and to supply proper occupation. This is particularly the case with those predisposed to tuberculosis, for those predisposed to rheumatic conditions, for those with heart lesions, with tendencies to skin disease, with catarrhal conditions and the like, which are so likely to be activated by unsuitable occupations. Individuals with flat chests and weak musculature must not work at indoor occupations, but must be trained for out of doors. The cardiac subject must not engage in an occupation requiring muscular exertion; the rheumatic in exposed occupations; the catarrhal in dusty or superheated occupations; those with spinal curvature, where heavy lifting is required;

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(a) New York Med. Jour., Sept. 23, 1916.

those with flat feet in ambulant occupations, etc. Individuals with repulsive facial conditions can not engage in occupations which require personal contact with the public, even though they are physically and mentally competent. The bar is even greater than if they were afflicted with gross physical deformity or mental defect. They would be the very persons to do the intensive research work so necessary in many callings.

The possibility of teaching new occupations to those with serious acquired defects, who have as a result been shut out from their occupations, and who must choose new occupations from within restricted ranges, has been demonstrated in Europe as a result of the war.

In conclusion, Friedman emphasizes that in consideration of the vast number of defectives in every community, something more must be done for them than mere diagnosis and separation, or mild remedial measures, or custodial care and the like. These solve the problem for neither the defective nor the community. Defects must be ascertained, measured, vocational possibilities inquired into, senses and faculties trained especially in reference to life vocations. Finally, the tendency to relegate the physically defective to the shades of "innocuous desuetude" can best be overcome, he declares, by selected physical training, democratic military training, in order to develop the physical condition of the people generally, and of the defective population specially.

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